Monograph on **Dental Caries & Carbonhydrates** 4/11/75

H7

The Sugar Association, Inc.

1511 K Street, N. W. Washington, D. C. 20005

G. Norris Bollenback, Ph.D.
Scientific Director

April 11, 1975

Mr. Richard Ronk, Director
Division of Food and Color Additives
Bureau of Foods
Food and Drug Administration
200 C Street, SW
Washington, DC 20204

Reference: Monograph on Dental Caries and Carbohydrates; TR-72-1552-61

Dear Mr. Ronk:

I have studied the reference monograph and consider the coverage highly acceptable and the evaluations in the Summary, Animal Studies and Human Studies commendable in their objectivity.

Reproductions of several papers which, I believe, augment the lengthy bibliography in the monograph are enclosed. These are submitted not to confuse the caries issue nor to promote any particular aspect of it. They are primarily meant to emphasize what is underscored early in the monograph summary, namely that dental caries is a multifactorial disease.

The articles by Bibby (The Cariogenicity of Snack Foods and Confections, JADA 90, 121-132 [1975]) and Edgar, et al (Acid production in plaques after eating snacks: modifying factors in foods, JADA 90, 418 [1975]) update and objectively view the current attention being given which associates caries with between meal snacking. Then, to suggest that between meal eating is still lacking in total variables playing on causes of caries, there is included, the article by Bagramian and Russell (Epidemiologic Study of Dental Caries Experience and Between-Meal Eating Patterns, J. Dent. Res., 52, 342 [1973]), showing, as I read it, no association of caries incidence with between meal snacking.

An excerpt from the British Survey of Children's Dental Health in England and Wales 1973 also cautions against assigning a one-to-one relationship of caries with, e.g., consumption of biscuits and cakes.

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Mr. Richard Ronk
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There seems to be a strong tendency to associate wartime sugar rationing with lowered caries incidence. The article (in translation) by Wandelt (Statistische Erhebungen Zur Frage Der Beziehungen Zwischen Zuckerverbrauch und Zahncaries, Ernahrungs-Umschau 15, 302 [1968]) shows there is a strong difference of opinion on this point, too.

As a final offering, the Carlsson and Johansson paper, Sugar and the Production of Bacteria in the Human Mouth (Caries Res., 7, 273 [1973]) examines the oral microflora. An important point brought out here is one that many overlook, namely, that micro-organisms need other than an energy supply in order of proliferate. Whether nutrients, such as minerals and vitamins, serving the microorganisms originate in the oral fluid (as proposed in the article) or in ingested foods is a detail subsidiary to the recognition that such nutrients are required.

In this same category, I might add that trace elements within the tooth may discourage (e.g., fluorides) or encourage (e.g., selenium) caries development.

Other articles enclosed include three that add to those cited in the monograph dealing with cereal products. These are 1) Brewer, et al, A clinical study concerning the Anticariogenic effects of NaH_2PO_4--enriched breakfast cereals in institutionalized subjects: results after two years, JADA, 80, 121 (1970); 2) Glass and Fleisch, Diet and dental caries: dental caries incidence and the consumption of ready-to-eat cereals, JADA 88, 807 (1974); 3) Peterson, North Dakota Field Test of Cariostatic Effect of 1% Sodium Dihydrogen Phosphate and Disodium Hydrogen Phosphate Added to Presweetened Breakfast Cereals, J. Dent. Res., date?

Miscellaneous articles and several IADR Abstracts (1973-1974) round out the submissions.

I trust these publications will be helpful to the Select Committee members in their evaluations.

Sincerely,

G N Bollenback

G. Norris Bollenback, Ph.D.
Scientific Director

GNB:db

enclosures

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The Role of Diet Surveillance in Water Fluoridation Programs. E. POWELL* and G. TURIANO. Univ. of Conn. and (formerly) Eastman Dental Center, Rochester, N.Y.

Water fluoridation studies show considerable variation in the level of reduction of dental caries. Although diet-dental caries relationship is well established, "diet surveillance" is not routinely done. The purpose of this study was to investigate diet patterns of children in fluoridated and unfluoridated communities in order to yield information for an appraisal of dietary factors in relation to this variance. Ninety-seven high school children living in a community with fluoridated water and 125 children of similar age from an unfluoridated area were selected for the study. Data were collected by self-administered questionnaires; results were tabulated and scored according to: food sugar content, oral clearance rate and frequency of food intake. Student group scores for these three diet categories were compared with "Model Diet Scores" previously determined. Findings in this study showed that while diet scores for both groups exceeded the model, student scores from the fluoridated community were consistently higher in magnitude, thus more cariogenic, than similar scores for the unfluoridated area students. Mean "model scores" were: (a) total score 42, (b) sugar content 16 and (c) oral clearance 8. Fluoridated and unfluoridated student groups respectively yielded mean scores of: (a) total score 49.15 and 44.99 (b) sugar content 21.15 and 18.30 and (c) oral clearance 11.30 and 10.40. These results were significant ($p<.05$). The mean eating frequency scores for the two student groups were not significantly different. These findings suggest diet surveillance as a useful procedural dimension for appraising the variance reported in water fluoridation programs.

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Oral Retention of Carbohydrate Foods
J. E. ROWLEY*, B.G. BIBBY and S. MUNDORFF
Eastman Dental Center, Rochester, N.Y. 14603

To add more definition to the role of different carbohydrate foods in caries causation, measurements were made of the amounts remaining in the mouth after eating. Fifty-four snack-type foods were tested in three human subjects who had dissimilar dentitions. Samples for analysis were collected 5, 15 and 30 minutes after normal ingestion of measured aliquots of the foods. Collection procedure consisted of pooling (a) mouth rinsings with 15 ml of water for 15 sec. (b) mouth fluids from toothbrushing with a wet brush (c) a second rinsing with 15 ml of water and (d) toothbrush washings. After appropriate dilution, anthrone in ethyl acetate and H_2SO_4 were used to produce the green carbohydrate color reaction. Aliquots that would fall within a 20 to 60% transmission range were measured on a Bausch and Lomb Spectronic Colorimeter and a regression equation used to calculate the carbohydrate content as glucose equivalents.
The 3 test subjects generally gave consistently high, medium or low retentions in food tests. The means for the 3 subjects showed wide ranges between foods at each time period. At 5 min. the highest mean retention was more than 20 times that of the lowest sugar containing snack. Mean retentions of all foods at 5 min. were approximately 3 times those at 15 min. and the latter twice those at 30 min. At the 30 min. period there was less than a 3-fold difference between the highest and the lowest retention and the highest carbohydrate level was less than 4 times that of the control saliva.

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Cereal Cariogenicity as Related to Sugar and Nutritional Content. G.K. Stookey* and J.L. McDonald, Jr., Oral Health Research Institute, Indianapolis, Indiana.

The purpose of this two-part study was: 1. to determine the cariogenicity of two different physical forms of sucrose in breakfast cereals; and 2. to compare the cariogenicity of various commercially available breakfast cereals. In Part I, 4 groups of rats were provided, respectively, diets containing either a dried cereal base, a sugar-coated cereal, or a cereal with one of two different levels of added sugar. In Part II, 11 groups of rats were provided different breakfast cereals which varied in their inherent sucrose and nutritional content. Both series were also run in hamsters. The findings in Part I showed that there were no significant differences in caries scores, in spite of the differences in physical form and quantity of sucrose ingested. In Part II, the caries-producing potential of the various cereals could not be directly related to their sugar content. For example, the lowest caries scores were observed in two groups provided "high nutrition" type cereals only one of which was pre-sweetened. Similarly, of the two products causing the highest caries scores, only one was pre-sweetened. Inherent factors other than sucrose content and relative tackiness, play important roles in determining the cariogenicity of breakfast cereals.

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Plaque pH Changes After Snack Food Use.
W. M. EDGAR and B. G. BIBBY*, Eastman Dental Center, Rochester, New York.

The effect of consumption of 59 snack foodstuffs on the pH of plaque was assessed in five subjects for each foodstuff. Plaque was sampled before, and at 0, 5, 10, 15, 20 and 30 minutes after food use. The pH was determined extra-orally using a one-drop electrode assembly. Saliva flow rate and pH were estimated during the experiment by the collection of 1 minute 'drooled' or expectorated samples of oral fluids between plaque sampling. Mean plaque pH minima and the duration of the pH depressions were compared in a two-way analysis of variance which indicated significant differences between plaque reactions to different snacks. When arrayed in order of plaque pH minima the foods fell into six groups in ascending acid-provoking potential. Reasons for the ranking of foods within individual groups are being sought in their carbohydrate content (qualitative and quantitative), pH and buffering power stimulus to salivation, and oral retention times. It is believed that these and other tests on foods will yield information as to their relative cariogenicity.

This section of the Journal of Dental Research *is devoted to rapidly published short research notes*

North Dakota Field Test of Cariostatic Effect of 1% Sodium Dihydrogen Phosphate and Disodium Hydrogen Phosphate Added to Presweetened Breakfast Cereals

JOHN K. PETERSON

North Dakota State Department of Health, Bismarck, North Dakota 58501

Favorable cariostatic results have been reported with the addition of a 1% sodium dihydrogen phosphate and disodium hydrogen phosphate mixture to breakfast cereals eaten by children (G. STOOKEY, R. CARROLL, and J. MUHLER, *JADA* 74:752, 1967), and adults (R. CARROLL, G. STOOKEY, and J. MUHLER, *JADA* 76:564, 1968).

Four hundred and twenty-one children, grades three to eight, in Grafton, North Dakota participated in a two-year test in which they ate seven presweetened breakfast cereals that contained the same additive used in the aforementioned tests. All children received visual and radiographic dental examinations. They were classified according to DMFS prevalence, dental age, and family and then assigned randomly to two groups. One group received cereal with the phosphate added; the control group received the same cereals without the added phosphate.

The table shows the mean caries increments measured in this study.

In an attempt to determine why these results differed from the previously mentioned studies, separate analyses were made of visual examination results alone and of results from blind retrospective examination of mixed baseline and final films. Separate analyses were made for all posterior proximal surfaces and for those surfaces fully readable on both examinations.

No cariostatic effect from the addition of the phosphates to presweetened breakfast cereals was demonstrated by any of these methods of analysis.

This investigation was supported in part by the General Foods Corporation.

Additional information available on request to author.

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TABLE

TWO YEAR CARIES INCREMENT (CEREAL STUDY)

Group	No. of Children	New DMFT		New Proximal Surfaces		Total New DMF Surfaces	
		Mean	SE*	Mean	SE	Mean	SE
Study	202	4.2	0.2	6.0	0.4	9.0	0.5
Control	219	3.8	0.2	5.2	0.3	8.0	0.4
t value	...	1.5	...	1.7	...	1.5	...

* SE, standard error.

Jan. 1970

clinical study concerning the anticariogenic ffects of NaH_2PO_4-enriched breakfast cereals in stitutionalized subjects: results after two years

rold E. Brewer, DDS, PhD
rge K. Stookey, MSD
seph C. Muhler, DDS, PhD, Indianapolis

o comparable groups of patients in an institution eived 1-oz servings of ready-to-eat cereals daily two years. One group received NaH_2PO_4-enriched eal. Dental examinations before, during, and at conclusion of the study showed that those re- ving the enriched cereal had reductions in den- caries increments approaching 50%.

al studies concerning the clinical efficacy of ary phosphates in humans have been some- t contradictory, although more recent ones est that the addition of phosphate to the hu- diet may be associated with the prevention of al caries. Although Stralfors[1] reported a sig- nt anticariogenic effect associated with the ion of dicalcium phosphate to the diet of children, subsequent studies by independent stigators[2-4] failed to confirm these observa- s. More recent reports[4-6] have indicated that icant reductions in the incidence of dental s in both children and adults are associated with the addition of NaH_2PO_4 to the diet in ready-to-eat breakfast cereals. The results of studies reported by Finn and Jamison[7] and Harris and co-workers[8,9] have confirmed the effective- ness of phosphate supplementation in humans with regard to the prevention of dental caries.

In previous studies at Indiana University[4-6] families were provided with the NaH_2PO_4-en- riched breakfast cereals and no attempt was made to modify or control their use of the cereals. It was subsequently decided to investigate the effec- tiveness of this nutritional approach to dental caries control under conditions whereby ingestion of the cereal was on a regular daily and supervised basis.

Method

A total of 474 subjects living in an institution for the mentally retarded participated in this program. The subjects were given a thorough clinical exam- ination by a single examiner (HEB) at the initia- tion of the study. They were distributed randomly into two experimental groups according to pro- cedures previously described.[10,11] All subjects were given a 1-oz serving of the respective ready- to-eat cereals daily by attendants at the institution. The cereals were provided in plain white, coded packages; attendants and supervisors were respon- sible for giving the appropriate cereal to each sub-

Table 1 ■ Range in age of subjects.

All subjects present at	Age of subjects at initial examination											
	4	5-9	10-14	15-19	20-24	25-29	30-34	35-40	40-44	45-49	50-+	Total
Initial examination												
Group 1	2	21	52	65	29	18	15	12	9	5	7	235
2	0	24	51	61	37	20	17	15	6	4	4	239
6-month exam												
Group 1	2	20	44	56	28	16	14	11	9	5	7	212
2	0	22	46	53	29	19	16	12	6	4	4	211
12-month exam												
Group 1	2	20	40	54	23	13	12	11	7	4	6	192
2	0	21	46	49	24	15	14	12	5	4	4	194
18-month exam												
Group 1	2	20	41	51	22	14	8	10	6	5	4	183
2	0	19	45	44	23	17	15	10	5	3	2	183
24-month exam												
Group 1	2	17	28	36	12	10	4	10	5	3	4	131
2	0	16	29	36	17	14	13	11	4	2	2	144

ject every morning of the study period. Records were maintained by the supervisors to show that each subject ingested the appropriate cereal daily. All of the meals for all of the subjects participating in the program were prepared in the same kitchen and delivered to the respective housing units where the subjects lived. All subjects participating in the program used a nonfluoride, non-tin-containing dentifrice; the drinking water of the institution was fluoride-deficient (less than 0.1 ppm F). Subjects who became hospitalized during the course of the project were given the proper cereal in the hospital. Any subject leaving the institution for a period of more than 30 days during the course of the project was dropped from the study.

After 6 months, 12 months, 18 months, and two years, all subjects were reexamined and radiographed by the original examiner with use of identical examination technics. The radiographs were read and recorded independently by a second investigator.

Results

The data obtained in this study are summarized in Tables 1 to 3. The range in age of the subjects initially as well as at each of the subsequent examinations is shown in Table 1. Although a wide age range existed, the distribution of the subjects into the two groups was comparable for all age groups throughout the study period.

Table 2 summarizes the balance of the subjects present at the initial examination, as well as at each subsequent examination, expressed in terms of DMF teeth and DMF surfaces. The subjects in Group 1 had a mean value of 10.40 DMF teeth and 20.06 DMF surfaces at the initial examination and those in Group 2 had comparable values of 10.31 and 19.57 DMF teeth and surfaces, respectively. The data indicate that throughout the study period no evidence of imbalance occurred.

The results of the dental caries examinations are summarized in Table 3. At the six-month examination period the subjects who received the control cereals (Group 2) had mean dental caries increments of 2.95 DMF teeth and 5.47 DMF surfaces. For this same period the subjects who received the NaH_2PO_4-enriched cereals (Group 1) had dental caries increments of 2.19 and 3.33 DMF teeth and surfaces, respectively. A comparison of the values of the two groups indicated that the subjects who received the phosphate-enriched cereals had reductions in dental caries increments of 29.2% and 39.1% for DMF teeth and surfaces, respectively. The results obtained at the subsequent examinations indicated that slightly smaller differences between the two groups were observed at the 12-month examinations than at the 6-month examinations, although the difference between the experimental and control groups continued to be statistically significant ($P<0.01$). The results obtained at the 18- and 24-month examinations were substantially improved as compared to the results obtained after 6 months. The occurrence of smaller increments in both groups after 18 and 24 months as compared to the 12-month examination can only be explained on the basis of a random loss from both groups of subjects who had a higher caries susceptibility. Differences in dental caries increments after 18 months indicated that the subjects who received the phosphate-enriched cereals had dental caries reductions of 28.6% and 46.1% for DMF teeth and surfaces, respectively, whereas reductions of 48.5% and 55.3% were observed after 24 months. All of the dental caries reductions were statistically significant. An examination of the findings on the interproximal surfaces indicates that reductions ranging from 82.1% to 97.4% were observed in the incidence of new interproximal lesions during the course of the study.

Table 4 summarizes the 24-month dental caries increment data tabulated according to the age of the subject. These data indicate that the anticariogenic effect of the phosphate-enriched cereals is not age related and that the cereals reduced the incidence of dental caries in all ages of subjects participating in the study.

Discussion

The data presented with regard to initial balance and distribution of age of the subjects who participated in this study indicate that not only was the balancing procedure quite effective in stratifying the subjects into two comparable groups, but it also was effective in maintaining the balance of these groups throughout the study period. Comparable observations have been made earlier with use of this same procedure to establish initial balance and stratify the subjects randomly into the various experimental groups.

The results of the dental caries examinations confirm previous observations on the value of NaH_2PO_4 in reducing the incidence of dental caries in humans. The results of previous studies in which NaH_2PO_4-enriched ready-to-eat breakfast cereals were given to children and adults without any attempt to modify or control consumption showed dental caries reductions ranging from 20% to 40%. The reductions observed in DMF surfaces during the first year of this study paralleled those observed in previous investigations. However, as the study progressed, the degree of protection expressed in terms of DMF surfaces tended to increase; a dental caries reduction of 55.3% was observed after two years. As compared to the previous studies in which no such tendency of increased effectiveness was observed, these data would suggest that continued use of the NaH_2PO_4-enriched cereals on a regular basis increases the prevention of new carious lesions. Attempts to ascertain the level of cereal consumption in the previous studies indicated that during the early part of these studies consumption was approaching a 1-oz serving daily, whereas during the second year of these projects consumption was some-

Table 2 • Summary of data for subjects present at each examination.

All subjects present at:	No. of subjects	Average at initial examination DMFT	DMFS
Initial exam			
Group 1	235	10.40	20.06
2	239	10.31	19.57
6-month exam			
Group 1	212	10.72	20.75
2	211	10.35	19.60
12-month exam			
Group 1	192	10.25	19.78
2	194	10.03	18.92
18-month exam			
Group 1	183	10.19	19.50
2	183	9.96	18.75
24-month exam			
Group 1	131	9.94	19.10
2	144	9.96	18.93

Table 3 • Summary of dental caries results for all subjects who completed the specified portion of the study.

Group and cereal	No. of subjects	DMFT Mean increment	DMFT % reduction	DMFS Mean increment	DMFS % reduction	Interproximal Surfaces Mean increment	Interproximal Surfaces % reduction
6-month data							
1. Presweet + agent	212	2.09	29.2**	3.33	39.1**	0.06	97.4**
2. Presweet control	211	2.95		5.47		2.34	
12-month data							
1. Presweet + agent	192	4.53	16.7*	7.41	29.2**	0.72	82.1**
2. Presweet control	194	5.44		10.46		4.03	
18-month data							
1. Presweet + agent	183	3.45	28.6**	5.33	46.1**	0.68	86.4**
2. Presweet control	183	4.83		9.89		4.99	
24-month data							
1. Presweet + agent	131	2.42	48.5**	4.19	55.3**	0.15	96.9**
2. Presweet control	144	4.70		9.38		4.87	

* Differences significant at < 0.01 level of confidence.
** Differences significant at < 0.001 level of confidence.

Table 4 ■ Summary of dental caries increments after two years according to initial age of the subjects.

Group and cereal	Age range (yrs)	No. of subjects	DMFT Mean increment	DMFT % reduction	DMFS Mean increment	DMFS % reduction	Interproximal Surfaces Mean increment	Interproximal Surfaces % reduction
1. Presweet + agent	<9	19	1.89	47.9	3.32	36.0	0.05	107.9
2. Presweet control		16	3.63		5.19		0.63	
1. Presweet + agent	10-19	64	3.08	37.4***	4.75	42.8***	0.28	92.8***
2. Presweet control		65	4.92		8.31		3.88	
1. Presweet + agent	20-29	22	2.32	54.8**	4.14	68.3***	-0.23	103.4***
2. Presweet control		31	5.13		13.06		6.71	
1. Presweet + agent	30-39	14	1.36	71.8**	3.21	69.4***	0.50	93.8***
2. Presweet control		24	4.83		10.50		8.00	
1. Presweet + agent	>40	12	1.17	61.0	3.83	56.2*	0.08	98.4**
2. Presweet control		8	3.00		8.75		4.88	

* Probability <0.05.
** Probability <0.01.
*** Probability <0.001.

what reduced and approached the nationwide average of 3.0 servings per week.[12] Under such circumstances dental caries reductions were observed to be nearly constant with a slight tendency to decrease during the study period. The findings of this study suggest that if regular ingestion of the cereal occurs on a daily basis one may expect dental caries reductions approaching 50%.

The results obtained in this study, expressed as a function of the age of the subjects, confirm previous findings that indicated a significant reduction in the incidence of dental caries in both children[4,6] and adults.[5] In this study reductions in DMF surface increments after two years were 36.0% to 42.8% for persons less than 20 years of age and 56.2% to 69.4% for older participants.

Conclusions

A clinical study was conducted in 474 institutionalized subjects to determine the anticariogenic effect of NaH_2PO_4-enriched ready-to-eat breakfast cereals in institutionalized subjects who had the same diet and whose cereal consumption patterns were controlled. The results of this study after two years confirm earlier observations that reduction in the incidence of dental caries in both children and adults is associated with the ingestion of NaH_2PO_4-enriched cereal. In this study reductions of 48.5% and 55.3% in the incidence of dental caries in DMF teeth and surfaces, respectively, were observed.

This study was supported, in part, by a research grant from the Post Division, General Foods Corporation, Battle Creek, Mich.

Doctors Brewer, Stookey, and Muhler are in the Preventive Dentistry Research Institute, Indiana University Medical Center, 410 Beauty Ave, Indianapolis, 46202.

1. Stralfors, A. Effect of calcium phosphate on dental caries in school children. J Dent Res 43:1137 Nov-Dec 1964.
2. Ship, I.I., and Mickelsen, O. Effects of calcium acid phosphate on dental caries in children: a controlled clinical trial. J Dent Res 43:1144 Nov-Dec 1964.
3. Averill, H.M., and Bibby, B.G. Clinical test of addition of phosphates to the diet of children. J Dent Res 43:1150 Nov-Dec 1964.
4. Stookey, G.K.; Carroll, R.A.; and Muhler, J.C. The clinical effectiveness of phosphate-enriched breakfast cereals on the incidence of dental caries in children: results after two years. JADA 74:752 March 1967.
5. Carroll, R.A.; Stookey, G.K.; and Muhler, J.C. The clinical effectiveness of phosphate-enriched cereals on dental caries in adults: results after one year. JADA 76:564 March 1968.
6. Muhler, J.C., and others. A clinical study concerning the effect of different routes of administration of NaH_2PO_4 in reducing the incidence of dental caries in children. Unpublished report.
7. Finn, S.B., and Jamison, H.C. The effect of a dicalcium phosphate chewing gum on caries incidence in children: 30-month results. JADA 74:987 April 1967.
8. Harris, R., and others. Observations on the cariostatic effect of calcium sucrose phosphate in a group of children aged 5-7 years: a preliminary report. Aust Dent J 12:105 April 1967.
9. Harris, R., and others. The cariostatic effect of calcium sucrose phosphate in a group of children aged 5-17 years. Aust Dent J 13:32 Feb 1968.
10. Muhler, J.C. Effect of a stannous fluoride dentifrice on caries reduction in children during a three-year study period. JADA 64:216 Feb 1962.
11. Muhler, J.C. Ability of different clinical examination techniques to diagnose caries prevalence. J Dent Child 30:3 1st quart 1963.
12. Market Research Corp. of America. Third annual household menu census. Quarterly report, July-Sept 1967.

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Enamel Protective Factors in Foodstuffs. M. E. Weiss*, B. H. Clarkson and H. Berlin† Eastman Dental Center, Rochester, New York; † Lewistown, Pennsylvania.

Three methods: (1) half tooth decalcification; (2) solubility of radioactive enamel in bacterial fermentation products; and (3) an enamel surface "window" technique, were used to test extracts of plant materials for effects on enamel solubility. Using (1) it was found that 15 plant extracts offered a measure of protection against enamel dissolution in pH 4 lactic acid. With (2) solubility reductions of more than 90 per cent were obtained with several herb products. With (3) levels of enamel protection were lower but quite definite.
Tests designed to indicate how the extracts modified enamel solubility showed both organic and inorganic agents were involved; in method (2) inhibition of bacterial activity played a large part, and that in (3) where bacterial activity was not involved the enamel protection depended partly on inorganic (buffering) agents and the action of some organic components.

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Suppression of dental caries by chemical activation of the hypothalamic-parotid endocrine axis. R. R. STEINMAN* and J. LEONORA, Loma Linda University, Loma Linda, California.

Preliminary evidence suggests that the direction of fluid movement through the dentin plays a major role in resistance to dental caries. Fluid movement in the dentin is hormonally controlled by the hypothalamic-parotid gland axis. Components of the ornithine cycle, particularly urea, stimulate the hypothalamic-parotid axis which activates fluid movement through the odontoblastic processes in the dentin. The present study was conducted to determine whether the continuous chemical activation of the hypothalamic-parotid axis with subcutanuous (sc) administration of exogenous urea or citrulline would decrease the incidence of dental caries in rats maintained on a high sucrose cariogenic diet for 13 weeks. Seventy-six male Sprague-Dawley 21 day old rats were divided into five groups and treated as follows: Group I twenty rats saline sc 3 times a day; Group II seventeen rats 20 mg urea sc/100 gm body wt/2 times a day; Group III thirteen rats 80 mg urea sc/100 gm 3 times a day; Group IV twelve rats 240 mg urea sc/100 gm 3 times a day; Group V fourteen rats 60 mg citrulline sc/100 gm 2 times a day. The number of cavities after 13 weeks was 15.6 ± 1.7; 9.9 ± 1.3; 4.5 ± 1.3; 1.8 ± 0.3; and 11.2 ± 1.5 respectively. All animals were autopsied. The animals receiving 240 mg urea 3 times a day showed evidence of stress having enlarged adrenals, spleen and kidneys and lower weight than the control animals. Those receiving 80 mg urea 3 times a day or less showed little or no evidence of stress.

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The Relative Effect on Dental Caries of Three Food Supplements to the Diet. Sidney B. Finn*, Homer Jamison, Institute of Dental Research, University of Alabama in Birmingham, School of Dentistry, University of Missouri, Kansas City, Missouri.

Over 600 children between 6 and 18 years of age in residence for 9 months of the year at a state institution, were divided into three equal groups of over 200 each. The three dietary additives were fed at the breakfast meal. All children consumed the same basic diet. Supplement one was a sugar-coated cereal; supplement two, raisins and fruit juices, and supplement three, a non sugar-coated cereal containing approximately 0.4% disodium phosphate incorporated into the cereal during the processing. Sugar and other sweets and carbohydrates in their daily diets were not restricted at any meal. After 18 months on these regimes, examinations with mouth mirror and explorer, and bite-wing radiographs revealed the following results. The mean DMF teeth of regime one was 1.57, regime two was 1.43 and regime three was 1.44. The mean DMF surfaces in regime one were 3.13, regime two 2.61 and regime three 2.99. Statistical analysis of these differences did not approach significant levels. Determinations based on the number of available surfaces did not reveal any significant differences either. This was a double blind study. The results would indicate that under the conditions of this study a sugar-coated cereal does not produce a significant change in dental caries incidence when compared to uncoated cereals or fruits containing natural sugars when eaten once a day in an unrestricted carbohydrate diet.

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Clinical Investigation of Dual Alloy Restorations in Teeth with Deep Subgingival Caries ELENA L. LIATUKAS*, College of Dentistry, Howard University, Washington, D.C.

A two-year study of gold-to-amalgam fillings restoring deep subgingival caries was made. The subgingival "problem" areas of caries were located both proximally and bucally. Forty adult permanent teeth were included in the study. In each case, a cast gold restoration was indicated but would have been technically difficult to construct due to caries depth. The technique used involved the buildup of gingival floors above soft tissue height with Ag amalgam prior to construction of the cast gold fillings. Extreme care was taken to position the gold to amalgam margins above the gingival crest for ease of clinical evaluation. Saliva pH was measured at pre- and post-restoration intervals. Other post-insertion clinical evaluations included: corrosion, discoloration, pitting, metallic taste and other discomfort related to the restored teeth. No significant change in pH occurred between pre- and post-insertion measurements (range 6.8-7.1). Surface discoloration was seen in two cases only at the 6 month interval. Discoloration observed was easily removed with prophylactic paste. Metallic taste occurred in one patient only following placement; however, spontaneous disappearance occurred after 1 month. No clinically significant changes in surface texture, marginal adaptation or corrosion were found in the cases tested. Microevaluations of these properties are being conducted.

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Comparative Dental Caries Activity in Dropouts and Non-Dropouts from a Three-Year Study

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Design of a clinical study to determine the impact of an experimental agent or regimen on dental caries activity must balance a number of interacting variables to yield meaningful results. At the outset, caries experience must be determined for each subject to permit sample stratification that will, in addition to age and sex, divide dental caries risk equally between the experimental groups. Unfortunately, some individuals discontinue participation during the course of the study. How damaging this will be to group caries risk equality depends on several factors, some obvious and others inapparent. For example, a correlation might exist between those habits or attitudes that are expressed overtly in discontinuance of participation and the measure under examination—dental caries activity. Could it be that dropouts have a peculiar or unusual dental caries rate or risk, and, by withdrawing unequally from experimental groups, do they destroy otherwise legitimate sample comparability? This study examines comparability of dental caries experience during a one-year period in two study samples; one group subsequently discontinued participation and the other completed all three years of the study.

This study was sponsored by The University of Michigan, financially supported by General Mills, Inc., Minneapolis, Minn, and conducted with the cooperation of the Ann Arbor Public Schools System.

Examiners during the field study were J. A. Regezi, captain USAF, DC, Travis Air Force Base, Calif and J. Philip Sapp, Department of Pathology, The University of Western Ontario, London, Ontario, Canada.

Received for publication July 24, 1972.

Ann Arbor seventh grade public school children of both sexes were enrolled into a permissive dental caries prevention study by parental consent. Dental caries activity was determined initially and annually thereafter. Examinations were conducted by two experienced dentists. One conducted all clinical examinations; the other conducted all radiographic examinations at the 0, 12, 24, and 36 month intervals. Participants were instructed individually in oral hygiene by a hygienist; this was followed by supervised brushing. The participant was seated in a head-rest-equipped portable dental chair and teeth were examined with a mirror and explorer. High intensity light was carried uniformly to each of the individual's teeth by a fiber optic light catheter within a hollow-handled mouth mirror. Verbalized observations were recorded on IBM forms (by another dentist). Seven X rays (bitewing and anterior) were taken per individual per year. Of the participants, 544 were examined at both the initial and one-year anniversary. Those present at examinations one and two, but who subsequently discontinued participation, comprised the dropout sample (188 individuals). The remaining 356 individuals continued participation for three years.

The table indicates the similarity between the two groups. No statistically significant differences were found between groups in initial, final, or incremental DMF teeth or DMF surfaces.

Discontinuance of participation thus was unrelated to caries activity.

DENTAL CARIES ACTIVITY IN DROPOUTS AND NON-DROPOUTS

	Dropouts (188) Mean	Participants (356) Mean	Dropouts SD*	Participants SD	Difference	Percent	t
Age (in years)	13.14	13.09	0.43	0.38	−0.05	0.39	−1.36
Sex	0.54†	0.51†	0.50	0.50	−0.03	−6.13	−0.69
Initial DMF teeth	4.74	4.59	3.36	2.89	−0.15	−3.31	−0.53
Initial DMF surfaces	6.61	6.26	5.15	4.60	−0.35	−5.51	−0.77
Final DMF teeth	5.73	5.71	3.91	3.33	−0.02	−0.37	−0.06
Final DMF surfaces	8.01	7.60	6.40	5.27	−0.40	−5.28	−0.74
Increments DMF teeth	1.51	1.59	1.54	1.58	0.08	4.82	0.55
Increments DMF surfaces	2.55	2.35	2.72	2.42	−0.20	−8.72	−0.87

* SD, standard deviation.
† Male = 1; female = 0.

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Myotatic Reflex Response to Unloading and Loading Jaw-Jerk Stimulation. D. C. McNAMARA, P. F. CRANE,* W. D. McCALL JR., and M. M. ASH JR., Univ. of Mich. School of Dentistry, Ann Arbor, Michigan 48104.

The electromyographic silent period (SP) following the jaw-jerk reflex has been attributed to several sensory feedback contributions. The objective of this study was to investigate the contribution of "muscle tension and length" on SP duration.

EMG from anterior temporalis and masseter, and vertical biting load of nine subjects with normal occlusions were recorded and displayed. During controlled clench at 5, 10, and 15 kg., standardized unloading jaw-jerks were delivered to the chin, followed by jaw taps in an upward direction. The latter resulted in a momentary increase of load between the teeth. Similarly, at a constant load and varying vertical openings of 10, 20, and 30 mm., ten consecutive unloading-loading jaw taps were applied.

Based on the statistically significant difference ($p<.01$) between the SP durations for different forces, it was concluded that there is a decrease in SP duration with an increase in force. The difference related to the type of stimulation was not statistically significant ($p>.05$). There was no statistically significant difference ($p>.05$) in SP duration related to different muscle lengths.

Supported by USPHS Grant DE 02731-07.

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Relative Effects of Polyphosphates on Parameters Possibly Related to Dental Caries. C. McGAUGHEY* and E. C. STOWELL, V. A. Hospital, Long Beach, California.

Pyrophosphate (PP), tripolyphosphate (TPP), trimetaphosphate (TMP) and hexametaphosphate (HMP) are known to reduce caries in animals. These experiments were done in an attempt to determine why TMP is superior to the others. Hydroxyapatite (HA) powder was stirred with equimolar (10^{-2}M) or equiphosphate (10^{-2}M PP) polyphosphate solutions (pH 7) for 15-30 min, the samples centrifuged and the HA washed with H_2O. The HA was treated with 5 ml of 5% HAc until near solubility equilibrium. The relative solubility reducing effects were: HMP > TPP > PP >> TMP. In other experiments, the treated HA was stirred with a metastable Ca (1.0 mM) and PO_4 (3.6 mM) solution (salivary concentrations, pH 7) for 5 min or allowed to stand with intermittent stirring for 2 days, and the amount of Ca and/or PO_4 bound was determined. Preliminary data suggest that inhibition of PO_4 binding was appreciably greater than that of Ca binding for all compounds, suggesting that they increase the Ca:PO_4 ratio of the bound mineral. The order of effects in inhibiting long term PO_4 binding and for increasing Ca:PO_4 ratio was (on equiphosphate basis) HMP > TPP > PP >> TMP. The first 3 compounds were nearly equal in inhibiting short term PO_4 binding. All compounds inhibited the adsorption of salivary proteins of both high and low adsorbability by HA, and the order of effects (on equimolar basis) was HMP ~ TPP ~ PP >> TMP (similar to short term PO_4 binding). It may be that the polyphosphate mediated increases in Ca:PO_4 ratio are the result of increases in the binding of Ca salts other than phosphate. If confirmed, this might explain the caries inhibitory superiority of TMP.

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The Intraoral Cariogenicity Test (ICT) in Young Subjects. C. A. Ostrom* and T. Koulourides, Institute of Dental Research, School of Dentistry, University of Alabama in Birmingham

Previous reports on the ICT (IADR 1973, Abs. 579, 580, 627) studied influences of selected factors on cariogenesis in adult subjects aged 35-60, who had numerous missing teeth and had relatively few remaining surfaces for natural caries. To extend validity of the ICT test, the Crozat appliance was adapted to carry the ICT test enamel slabs in acrylic flanges subjacent to the buccal surfaces of the mandibular molars. Three fully dentulous subjects, MH, SC, WH were aged 13, 15 and 23 and had caries experience in 10, 14, and 0 teeth. With no extraoral sugar supplementation, MH and SC showed low-moderate ICT cariogenesis; and WH showed none. With extraoral ICT 3% sucrose immersion for 4 daily 10 min intervals, MH and SC showed moderate and WH extremely low ICT cariogenesis. Four trials in each subject showed raffinose and sorbitol less cariogenic than sucrose, comparable to reported findings in older subjects.. Microbial counts taken 2 hours after pumice prophylaxis showed that the ICT Dacron gauze cover and the washed enamel slab were more similar to buccal plaque of 18 than to samples from the gingival crevice, vestibule, dorsum tongue or saliva. After the ICT had been worn 7 days with sucrose supplementation, microbial counts of the washed slab showed that Streptococcus sanguis adhered in large numbers and was more numerous than other viridans streptococci. This study showed ICT cariogenesis in young fully dentulous subjects similar to that reported in older subjects. This suggests that the ICT measures the current cariogenic activity in-vivo, independent of the host enamel.

Supported by Contract No. NIH-NIDR-72-2030.

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Sugar Cariogenicity on Experimental Caries in Humans. R. Bodden*, T. Koulourides, S. Keller, and L. Manson-Hing. Dept. of Oral Diagnosis and Inst. of Dental Res., Sch. of Dentistry, University of Alabama in Birmingham 35294

This study was designed to evaluate the contribution of fructose, glucose, maltose, mannitol, melibiose, raffinose, sorbitol, sucrose, and xylitol to cariogenicity as measured by an experimental intraoral model in humans (N. Y. Acad. Sci. 153(1): 84-101, 1968; IADR Abs. #827, 1973). Human volunteers with partial dentures carried enamel slabs covered with Dacron gauze, for enhancement of bacterial colonization, for one week per experiment. The sugars were provided to the plaques extraorally as supplements, 10 min 4 times daily. After one week in the mouth, the recovered slabs were evaluated for the degree of demineralization by measurement of indenter penetration. Concentrations of 1%, 3% or 10% sucrose approximately doubled the subject's cariogenicity attributable to the dietary substrates with no appreciable difference between the above 3 concentrations. Each sugar was tested against 3% sucrose, which we adopted as a standard for the comparisons. The results were computed in ratios of test sugar/sucrose. Ratios near or above 0.50 indicate virtual absence of cariogenicity in the test sugar, while those near 1.0 indicate cariogenicity equal to sucrose. With a minimum of 16 experiments per comparison, the ratios were: Sorbitol 0.73, raffinose 0.77, fructose 0.95, mannitol 0.71, melibiose 0.78, and xylitol 0.44. According to this data, xylitol would be the most promising sucrose substitute. With the exception of fructose, all other test sugars were less cariogenic than sucrose.

Supported by Contract No. NIH-NIDR-72-2030.

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The Clinical Effect of Phosphate-enriched Breakfast Cereals on Dental Caries Incidence In Adolescent Children. N.H. ROWE*, R. ANDERSON, L.A. WANNINGER. Sch. of Dent., Univ. of Mich., Ann Arbor and General Mills, Inc., Mpls.

...blic school 7th grade children of both sexes, aged 13 years, were enrolled into a permissive caries prevention study. Participants were segregated into 2 similar groups stratified by age, sex, race and previous caries experience. They were examined annually until age 16 years. Both groups received 7 varieties of ready-to-eat breakfast cereal ad lib during the study period. One group's cereal (experimental) contained 0.18% additional mono 3:1 disodium phosphate, the other (control) regular production cereals. Examinations were conducted by two experienced dentists; one conducted all clinical, the other all radiographic examinations. Participants' teeth were examined by mirror and explorer with high intensity light carried uniformly to each tooth by a fiber optic light catheter within a hollow handled mouth mirror. Verbalized observations were recorded on IBM forms. Radiographs were taken, 7/individual/year. Sample size at the beginning was 563 and at termination, 375 children. Incremental caries of teeth (DMFT) and surfaces (DMFS) of individuals in the two groups between exams 1 and 4 (3 years) revealed no statistically significant difference. Anterior teeth alone, posterior teeth alone, and posterior teeth excluding 1st permanent molars, all revealed the control group to have a slightly elevated caries incremental score, but not statistically significant. When only newly erupted teeth were compared, greater disparity between groups was observed but due to decreased sample size the difference was not statistically significant.

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Effect of $CaHPO_4$ and Trimetaphosphate (TMP) on Cariogenicity in Humans. T. KOULOURIDES*, T. HOUSCH, and H.C. JAMISON. Inst. Dent. Res., School of Dentistry, Univ. of Alabama in B'ham.

In previously reported studies using bovine enamel slabs worn for the Intraoral Cariogenicity Test (ICT) by human subjects, 3% sucrose supplementation for four 10 minute intervals daily approximately doubled the cariogenesis attributable to the host's dietary substrate. (N.Y. Acad. Sci. 153 (1):84-101, 1968; IADR Abs. #167, 1972). The purpose of the present work was to explore the effect of 1% $CaHPO_4$ or 1% sodium trimetaphosphate (TMP) additives on the cariogenicity of 3% sucrose supplements. Sixteen experiments were conducted for comparison of each additive against sucrose supplements. The degree of cariogenesis was evaluated from the differences between initial and final measurements of micro indenter penetration on presoftened and sound enamel. The micron penetration change ($\bar{X} \pm SE$) in ratios of sucrose + additive over sucrose alone for sound and presoftened enamel, respectively, were, $CaHPO_4$: 0.97±0.20/1.31±0.32; 0.49±0.21/0.96±0.37. TMP: 1.58±0.32/2.45±0.53; 1.07±0.31/1.82±0.52. These ICT results indicate considerable counteraction of the effect of supplemental sucrose by the additives. A subsequent test of acid resistance of the slabs recovered from the ICT showed no appreciable difference between the compared groups. In conclusion the two tested additives protect the enamel from cariogenic attack, but they have no residual effect on the resistance of enamel to future attacks in the absence of the additives.

Supported by Contract No. NIH-NIDR-72-2030.

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...ffering Capacity, Sugar Content, and Cario...icity of Foods. S. KATZ,* B. L. OLSON, J. L. McDONALD, JR. Indiana University School of Dentistry, Indianapolis, Indiana.

Observations conducted at our laboratories as well as elsewhere show that the cariogenic potential of different breakfast cereals, as measured both in animals and under mouth simulation conditions is not related to the sugar content of the cereal, nor to its retentiveness on the teeth. In fact cereals with high sugar content and high retentiveness have been observed to induce less caries than others rather low in sugar and with low retentiveness. The only parameter which appears to relate well with the caries-inducing ability of the cereals is their buffering (or acid neutralizing) ability. The buffering capacity of the cereals is due chiefly to their mineral content, which comes from two sources: those which are inherent in the cereal's ingredients and those which are added. In addition, some cereals are high in protein, and this also can contribute to buffering. We have been able to determine at least two kinds of buffering activity in cereals: bound or fixed, i.e., buffers that are not easily released; and soluble or readily available, which are released rather rapidly. The cereals which in our experiments, and those of others, induce the least caries are those rich in buffers in both categories. The behavior of different cereals as to buffering, sugar content, retention, and cariogenic potential is presented through tables and graphs, which are analyzed for their most meaningful conclusions.

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Correlation Between Cariogenic Indices of Foods and Rat Caries. B.M. SHRESTHA*, S.A. MUNDORFF, J. ROWLEY and B.G. BIBBY. Eastman Dental Center, Rochester, New York.

The König Automatic Rat Feeder was used to determine the relationship between the production of caries in rats and the cariogenic indices obtained for various snack foods by 'in vitro' decalcification from salivary fermentation, oral food clearance and plaque pH determinations.

Groups of 8-10 weanling albino rats were fed various test diets at the same frequencies for six week periods, at the end of which, all rats were sacrificed and their mandibular teeth examined and scored for caries using Keyes' method. Navia's #200 Cariogenic Diet was used as control and all rats were given distilled water 'ad libitum'.

Of the 8 snack foods tested, dark chocolate produced 39% more caries than the control group, while milk chocolate produced only 4% more. Although graham crackers and potato chips both produced less caries than the control, the grahams gave 15% more caries than the potato chips. Of the remaining four snack foods tested, fudge bar and coconut-chocolate bar with almonds produced 26% and 16% less caries, respectively, while caramels produced 4% more caries than the control. Coconut-chocolate bar without almonds gave approximately the same amount of caries as the control.

With the exception of the fudge bar, all the findings in rat caries correlated with those given by 'in vitro' cariogenicity indices.

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In Vitro Caries Production by Snack Foods
A.M. YAARI, F.O. BARRETT* and B.G. BIBBY
Eastman Dental Center, Rochester, New York.

An in vitro system (I.A.D.R. Abst. #139, 1973) that produces caries-like changes in enamel was developed for the study of the relative cariogenicity of various foods and for evaluation of plaque formation and maturation. In it tooth sections are exposed to an automatically controlled flow of thawing quick-frozen whole human saliva. Various carbohydrate foods, held on the tooth surface produced sub-surface enamel lesions, which, when viewed after 5 days, under polarized light, ranged from 10μ to 45μ in depth. Findings on the relative cariogenicity in this system, consistent with those given by in vitro and animal tests, include more extensive lesions with white bread (32u) than with brown bread (23u), and with dark chocolate (20μ) than with milk chocolate (10μ). Parallelism with in vitro enamel demineralization and oral food clearance tests has also been found with several other candies and cookies including gingersnaps (20μ), chocolate chip cookies (15u) and graham crackers (20μ). The extent of the "carious" lesions produced by additional foods already tested in animals and chosen because of the contrasting results obtained in in vitro laboratory tests, will also be presented.

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Programmed Feeding/Drinking System for Rat Caries Studies: Sucrose vs. Fructose.
D.P. CURRIE* and J.M. NAVIA. Institute of Dental Research, School of Dentistry, University of Alabama in Birmingham, 35294.

Studies on the caries potential of different sugars have yielded conflicting results. Control of feeding and drinking patterns, among other approaches, is essential to standardize caries testing in rats (Navia, J.M., J.Dent.Res. 49:1213, 1970). The purpose of this study was to develop a programmed feeding/drinking system to evaluate the cariogenicity of sucrose and fructose in solutions, thus avoiding the variability contributed by particle size of sugars supplemented in diets.

Forty-five male, weanling rats (SD-CRL) were randomly divided into 3 groups of 15 each and housed in special cages in which all animals received liquids and a solid diet at programmed intervals. All animals received a non-caries promoting basal diet (Gel-#453). The test groups drank 10% sucrose or 10% fructose solutions and control rats received distilled water. The liquids were offered 10 times and the diet 6 times during a 14 hour night period. To insure equal consumption of water, the volumes of test solutions offered contained equal amounts of water as that consumed by control rats. After 23 days the rats were sacrificed and the buccal, sulcal and proximal molar surfaces scored for caries (Keyes, 1958). Rats in the fructose group had significantly higher caries scores than those in the group receiving sucrose ($p<0.005$) in solution. Caries scores of rats drinking fructose or sucrose were significantly higher than those of rats drinking distilled water ($p<0.001$). The system proved to be a useful approach to test the caries potential of liquids containing sugars and food beverages. (Supported by NIH-NIDR Contract No. 72-2403 and DE-2670.)

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Caries Promoting Properties of Sucrose Substitutes in Foods: Mannitol, Xylitol and Sorbitol.
J.M. NAVIA*, H. LOPEZ, and J.S. FISCHER. Inst. Dental Res., Sch. of Dentistry, University of Alabama in Birmingham, 35294.

Mannitol (Mnl), xylitol (Xlt) and sorbitol (Sbl) have been suggested as sucrose substitutes in foods to reduce their caries potential. The object of this study was to evaluate the caries promoting properties of these sugar alcohols in comparison to those of sucrose (Suc) or fructose (Fru).

Four experiments were conducted with the following design: Groups of 32 weanling rats (SD-CRL) were fed a cornstarch diet #300 containing the test sugar (particle size:125-150μ) at a low level of 5%. After 20 days on this dietary regime, rats were sacrificed and the molars scored for buccal and sulcal caries by the method of Keyes (1958). Jams were prepared (Foremost Res. Center) replacing Suc with Xlt or a mixture of Fru and lactose. These jams were fed as snacks to rats in a programmed feeding cage (König, K., Arch.Oral Biol. 13.13, 1968) and after 20 days the rat molars were scored for buccal and sulcal caries.

Expt. #1 compared Suc, Fru and Sbl and showed no differences in buccal (10.5, 10.7, 10.3) or sulcal (10.4, 11.0, 8.7) scores. Expt. #2 compared Suc, Xlt and Mnl and also showed no significant differences in buccal (13.3, 15.1, 13.7) or sulcal (10.3, 10.6, 10.3) scores. Suc, Xlt and Mnl were retested in Expt. #3 with similar results: buccal (10.8, 10.5, 11.2), sulcal (9.5, 8.8, 9.5) scores. In Expt. #4, Suc jam was compared to Xlt and Fru-lactose jams and no reduction in buccal (3.1, 4.0, 4.2) or sulcal (9.7, 7.3, 11.0) caries was shown for substituted jams. Sugar alcohols, on the basis of animal tests, seem to offer no advantages as substitutes for sucrose in snack foods. (Supported by NIH-NIDR Contract No. 72-2403.)

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Rat Oral Flora Modifications Induced by Dietary Sugar Alcohols.
A.J. NARKATES* and J.M. NAVIA. Institute of Dental Research, School of Dentistry, University of Alabama in Birmingham, 35294.

Sugar alcohols, among other sweetening agents, have been suggested as sucrose substitutes in manufactured foods to reduce their caries potential. The object of this study was to investigate the modifications induced by xylitol or sorbitol in comparison to fructose, sucrose and starch in the oral flora of selected sites on molars of rats used to evaluate the caries potential of these sugars.

Male rats (SD-CRL), 19 days old, were distributed among 5 groups which were offered basal diet MIT #305 with the following modifications: I. 5% cornstarch, II. 5% sucrose, III. 5% fructose, IV. 5% xylitol, V. 5% sorbitol. On days 21, 30 and 41 after birth, rats were sacrificed and the buccal and sulcal surfaces of molars were selectively sampled and plated in differential media for streptococci, lactobacilli and coliforms. Streptococci were differentiated by colony morphology and fermentation patterns. Sulcal samples contained 100 times more colony forming units than buccal samples. Streptococci constituted 30-60% of the total counts. Buccal and sulcal flora from rats in groups IV and V were essentially all S. mutans. S. mutans isolated from rats in group V were of the serological type d, and fermented sorbitol and mannitol. The S. mutans isolated from group IV fermented xylitol as well as mannitol and sorbitol. Isolates from the other groups were mostly S. sanguis, S. mutans and a few coliforms and lactobacilli which diminished in number with increase in age. Dietary sugar alcohols induce a flora which is cariogenic and when tested in the rat model, show no advantages as sucrose substitutes in foods. (Supported by Contract No. 72-2405 and DE-2670.)

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...lative Cariogenicity of Natural and Refined ...gars. A.E. NIZEL*, S.A. MILLER, T.M. CLEARY, ...S. QUINN. Tufts, Boston, and M.I.T., Cambridge, Massachusetts.

The overall purpose of this study was to compare the cariogenicity of sucrose derived from natural food sources with that derived from refined products.

Four groups of 8 day old hamsters were fed diets containing 71% carbohydrate (test sugar + cellulose), 21% lactalbumin, 4% corn oil, 3% salt mix, and 1% vitamin mix. The physical texture of the diets separated them into two categories: powder group (powdered sugar), and viscous group (invert sugar, or honey, or raisins).

To equalize the oral flora, all animals received oral inoculations of streptomycin resistant strep mutans.

After 107 days on their respective diets the animals were sacrificed and the molars scored for caries. There was no statistically significant difference in caries incidence between the groups fed sucrose or invert sugar. Also the caries experiences of those animals fed a honey or a raisin diet or an invert sugar diet were the same.

This suggests that both viscous and powdered sucrose can produce the same caries incidence. Furthermore, the current fad of substituting natural sources of concentrated sugar, (such as honey or raisins), for refined sugars in order to minimize dental caries development is probably not valid.

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The Relationship of Caries, Hygiene and Diet in 453 Children. M.A. LOYD and A.S. RICHARDSON. Faculty of Dentistry, University of British Columbia, Vancouver 8, B.C.

The interrelationship of diet, oral hygiene and caries was evaluated. Of the sample size of 453, 232 children were in Grade I and 221 were in Grade VII. Five-day diet surveys were obtained, followed by clinical examinations for oral hygiene and caries. By means of disclosing solution, mouth mirror and light, three oral hygiene indices were recorded: 6 surfaces, 4 molar surfaces and one molar surface. Using mouth mirror, explorer and extraoral light, four indices of caries activity were recorded: DMFT/T and DMFS/S for first permanent molars, and for total permanent teeth. In the Grade I children, two additional indices were used for caries: dmft/t and dmfs/s for D's, E's and 6's. From the diet surveys, refined and total carbohydrate consumptions were calculated per day as between meals and totalled for that day. The five-day totals, which included one weekend, were recorded along with frequency of eating per day and the 5-day total frequency. Computer analysis revealed no correlation between carbohydrate consumption and caries or oral hygiene indices and caries, r = 0.03 - 0.24. There was a low correlation (0.16) between frequency of eating and caries in first permanent molars. The correlation within the three oral hygiene indices was 0.70 - 0.77 and within the caries indices 0.51 - 0.94. No relationship was established between oral hygiene indices as recorded in this study and caries. Caries did not appear to be related to carbohydrate consumption as recorded by this five-day diet survey.

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...mel Dissolution by Carbonated Beverages, Fruit Drinks, and Fruits. S.A. MUNDORFF* and B.G. BIBBY. Eastman Dental Center, Rochester, New York.

To evaluate enamel destruction by carbonated beverages, fruit drinks and fresh fruits, measurements were made of their pH, natural acidity (NA), carbohydrate (CHO) content and oral clearance rate; and using previous methods (IADR 1973), pH changes, titratable fermentation acid (FA) and enamel decalcification ratios were determined after incubation in whole saliva.

Of the regular carbonated beverages, Orange Crush decalcified the most enamel, and Coca-Cola, the least. This difference could not be explained on the basis of FA or NA. The non-sugared pops, Tab and Fresca, each decalcified only 1/3 the amount of enamel of the regular pops. Here, the rise in pH after incubation seems to be the determining factor, rather than NA. Carbonation produced little, if any, effect on pH and enamel dissolution.

Among the processed fruit juices, the lowest decalcification ratios were given by the apple, grape and prune juices. This observation seems related to their comparatively lower NA and FA values. Orange and grapefruit juices had the highest decalcification ratios. In general, while the juices were more acidic, they cleared faster, and decalcified less than their corresponding fresh fruit.

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Measuring Attitudes Toward Dentists and Dental Care. F.S. FURIMA*, K.J. ROGHMANN and H.J.V. GOLDBERG. Univ. of Rochester and A.L. Jordan Health Center, Rochester, New York.

Patient satisfaction is an important aspect of quality of care. This study devised an instrument to measure satisfaction with dental care among neighborhood health center users.

Based on a scale of satisfaction with medical care and answers to an open-ended survey question, 35 statements expressing satisfaction and dissatisfaction with dental care were formulated. 15 statements were eliminated in pretests. The paper and pencil administration was replaced by a card sort technique using a small box with five slots for degrees of agreement or disagreement. The scale with 10 positive and 10 negative items was administered to 208 patients and reached a reliability of r=.678. A parallel 24-item scale measuring satisfaction with medical care had a reliability of r=.632.

14 physicians and 14 dentists in the Health Network judged the degree of satisfaction or dissatisfaction expressed in the statements. They also predicted the average patient response. Both physicians and dentists anticipated more dissatisfaction than was actually expressed.

An item-analysis, a factor analysis and the Thurstone rating helped to propose a final 13-item scale of greater homogeneity and reliability for future use.

Funded in part by O.E.O. grant #20029.

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TO WHAT ... DENTAL RA... APPRAISA... TIST'S A... COMPONE... IN STAND... RUSSEL IN... RATING AN... ADOLESCEN... THIS STUD... AGED 12-1... REPRESENT... WHO WERE ... AGING AN... 2-3 HOUR ... HOSPITAL... DRAWN USI... EACH STRA... THE FULL ... RATINGS. ... SELF RAT... INDICATOR... TWICE AS ... (12%) THE ... SELF REPO... WITH DENT... 3. DENTIS... RELATED T... FREQUENCY ... ADOLESCEN... DECAYED T... COMPONENT ... RATING WA... (OHI-S)(c...

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Diet and dental caries: dental caries incidence and the consumption of ready-to-eat cereals

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The estimated amounts of regular and presweetened ready-to-eat cereals consumed by 949 children during two years were analyzed to determine the existence of an association between caries incidence and the consumption of these cereals. The average cereal consumption by type of cereal was estimated for each participant, and the children were classified as low, medium, or high consumers of cereals. Analysis of data on need for restorative treatment in permanent teeth and in permanent and deciduous teeth, by type and amount of cereal consumed, showed no association between caries incidence and cereal consumption. The cariogenicity of a food may be related to its consistency, the time of consumption, and the conditions under which it is eaten, as well as its sucrose content.

In view of attention focused recently in the United States on the sugar content of children's diets in relation to dental caries, it seemed worthwhile to analyze from this viewpoint, the records of a clinical trial designed to evaluate the cariostatic effect of 1% sodium dihydrogen phosphate added to ready-to-eat cereals, including presweetened cereals. The children participating in this study were provided with generous amounts of these cereals free of charge for two years. As no phosphate treatment effect on caries incidence was observed (unpublished data by Glass), a finding later corroborated by an independent study,[1] data for experimental groups could be combined for other types of analyses.

In the present study the amounts of cereals consumed, both regular and presweetened, were analyzed and their relationship to several measures of the caries incidence observed during the experimental period of two years was examined.

Materials and methods

A total of 1,199 children from 690 families participated in the clinical trial. At time of entry to the study, all children were between 7 and 11 years of age; they lived in eastern Massachusetts communities where the amounts of fluoride in the supplies of drinking water were not significant.

On entrance to the study, each participant was given an oral prophylaxis; comprehensive clinical and radiographic examinations of the teeth were made. Three examinations were carried out by the same dentist under standard conditions at annual intervals. Nutritional histories were made for the children through their mothers

ORDER BLANK: You may order any assortment of cereals shown below. Write the number of packages of each product you wish to order opposite the product name. The TOTAL number of packages in your order should be a multiple of FOUR (4, 8, 12, etc.).

B0040 DATE ____________

____ 01. CORN FLAKES	____ 08. PEP (WHEAT FLAKES)
____ 02. 40% BRAN FLAKES	____ 09. COCOA KRISPIES
____ 03. RAISIN BRAN	____ 10. SHREDDED WHEAT
____ 04. RICE KRISPIES	____ 11. SPECIAL K
____ 05. SUGAR POPS	____ 12. OKs (OAT CEREAL)
____ 06. SUGAR FROSTED FLAKES	____ 13. FROOT LOOPS
____ 07. SUGAR SMACKS	____ 14. FROSTED STARS

____ TOTAL NUMBER OF PACKAGES

Fig 1 ▪ Cereal order form showing types of cereal provided. All order forms were preprinted with participant's identification number, here B0040. Address for delivery station was printed on reverse side of form, with name and address of participant.

by trained nutritionists. Records were also made of the number and age of members of the immediate family. During the examination of a child the mother was instructed in the method of ordering supplies of ready-to-eat cereals that were provided free on request to all participants and their families. Fourteen types of cereal, eight regular and six presweetened, were made available to provide the variety considered necessary for continuing participation. All cereals were provided by the Kellogg Co. Mothers were asked to encourage their children to eat as much cereal as they liked, and at least one serving daily. All cereals were provided in the same style of packaging as that available on the open market.

Free dental care was offered to all participants at the Forsyth Dental Center to provide additional and continuing incentive to cooperate. The treatment philosophy was similar to that of a practicing pedodontist except orthodontics, prophylactic odontotomy, restoration of mobile deciduous teeth, and fluoride treatments were not provided.

The records of 979 children are included in the analyses for the present study. Although 1,011 of the original children were still participating when the study was terminated, the 979 participants included here met the additional conditions of having all their dental treatment provided under the supervision of the project director at the Forsyth Dental Center; also all the records of the families' cereal orders were available for the entire two-year span of participation. As no treatment effect due to the phosphate supplement was observed, data for children in the experimental and control groups were combined for the purpose of the present analyses.

A blank cereal order form and the types of cereal provided are shown in Figure 1. The amounts and types of cereal requested and shipped in each order, participant identification number, and date of order were punched into machine records cards. All cereal order cards were sorted on date of order and family identification number and the information then coded on magnetic tape. Varieties of regular and presweetened cereals were identified and totaled to provide the number of boxes of regular cereal, boxes of presweetened cereal, and total boxes of cereal delivered to each family. Annual totals of each of the latter quantities were divided by the number of family members, excluding children under age 1, to estimate the average cereal consumption by type for each participant. These figures were verified on an individual basis through the dietary histories taken by nutritionists.

Findings of clinical and radiographic dental examinations were originally recorded on optically scanned, precoded examination records. These were processed by machine that encoded findings into punch cards to provide the data base for the calculation of caries increments.

Caries increments are shown in terms of new decayed and/or filled (DF) surfaces. These are expressed as net new DF surfaces (observed minus changes in diagnosis), and net annual caries incidence rate per 100 surfaces at risk[2]: rate equals $sd+sf+ud+uf-(ds+fs)\,/\,ss+us+sd+sf+ud+uf$ multiplied by 100, where the first letter in any pair indicates the surface status at an examination, the second letter indicates the status at a subsequent examination, and *s* equals sound, *d* equals decayed, *f* equals filled, and *u* equals unerupted. This caries incidence rate adjusts for any possible differences in tooth surface populations at risk. In addition, those tooth surfaces requiring restoration, according to each child's dental treatment plans made at the times of the dental examinations, were coded and punched into cards along with appropriate identification. These cards were processed by computer to obtain for each child, on an annual basis, the number of permanent tooth surfaces requiring restoration and the combined number of permanent and deciduous tooth surfaces requiring restoration. Summary cards containing this information were prepared for statistical analyses by computer.

Several methods were used to analyze the data. The mean age-specific counts of DMF teeth observed at each examination for each age group were plotted for inspection, along with the corresponding age-specific counts for all Massachusetts children. The DMF counts for the Massachusetts population used for comparisons were determined in an independent survey of 8,934 children of the same age distribution. The latter survey was carried out by the Dental Division of the Massachusetts Department of Public Health.[3]

The data on cereal consumption were used as continuous scale measurements in regression analyses of cereal consumption on age, caries increments, and needs for restorative treatment. In addition, this scale was collapsed to an ordinal scale to circumvent the difficulty involved in the exact measurement of cereal consumption. By means of this technique, children were classified as low, high, and medium consumers of cereal. Low consumers were defined as the lower 27% of users, high consumers as the upper 27%, and medium consumers as the middle 46%. This procedure utilizes a modification of a technique developed and validated by Kelley.[4] For these three groups, statistical analyses were carried out by analysis of variance for these variables: age; annual counts of new decayed and/or filled surfaces; annual caries incidence rate; annual counts of permanent tooth surfaces requiring restoration; and annual counts of permanent and deciduous tooth surfaces requiring restoration.

Table 1 ■ Distribution of children by age and sex at the time of the first examination.

Age	Boys	Girls	Total
7	66	78	144
8	129	126	255
9	121	115	236
10	110	97	207
11	79	58	137
Total	505	474	979

Table 2 ■ Mean counts of DMF teeth by age and sex of 979 children at times of three examinations, each one year apart.

	Age	Boys Mean	Boys SD	Girls Mean	Girls SD	Total Mean	Total SD
First examination							
	7	2.20	1.77	2.23	1.79	2.22	1.78
	8	2.73	1.91	2.66	1.66	2.69	1.79
	9	3.13	1.87	3.70	1.69	3.41	1.80
	10	4.14	2.07	4.69	2.66	4.40	2.38
	11	5.34	2.82	5.03	3.14	5.21	2.95
Second examination							
	8	2.89	1.68	3.27	1.62	3.10	1.65
	9	3.60	1.98	3.51	1.72	3.56	1.85
	10	3.84	1.85	4.74	2.62	4.28	2.30
	11	5.18	2.74	5.96	3.53	5.55	3.15
	12	7.58	4.00	7.24	4.66	7.44	4.28
Third examination							
	9	4.02	1.56	4.40	1.81	4.22	1.71
	10	4.94	2.80	4.73	2.38	4.84	2.60
	11	5.62	3.48	7.26	4.12	6.42	3.88
	12	7.57	3.94	9.11	5.50	8.30	4.78
	13	11.04	4.75	9.55	4.95	10.41	4.87

Results

The distribution of the 979 children is shown by age and sex in Table 1. Their caries prevalence as characterized by mean age and sex-specific counts of DMF teeth is shown in Table 2. The mean age-specific DMF counts are plotted by age and examination in Figure 2, along with corresponding mean age-specific values for 8,934 children (one examination only) from representative communities in Massachusetts.

The caries prevalence of those in the study approximates that of the Massachusetts population. The growth curves resulting from the mean age-specific DMF counts from the three examinations of study participants increase at a rate similar to that of the Massachusetts population.

Analyses of cereal consumption in terms of boxes per child per year are shown in Table 3. On the average, consumption of regular varieties

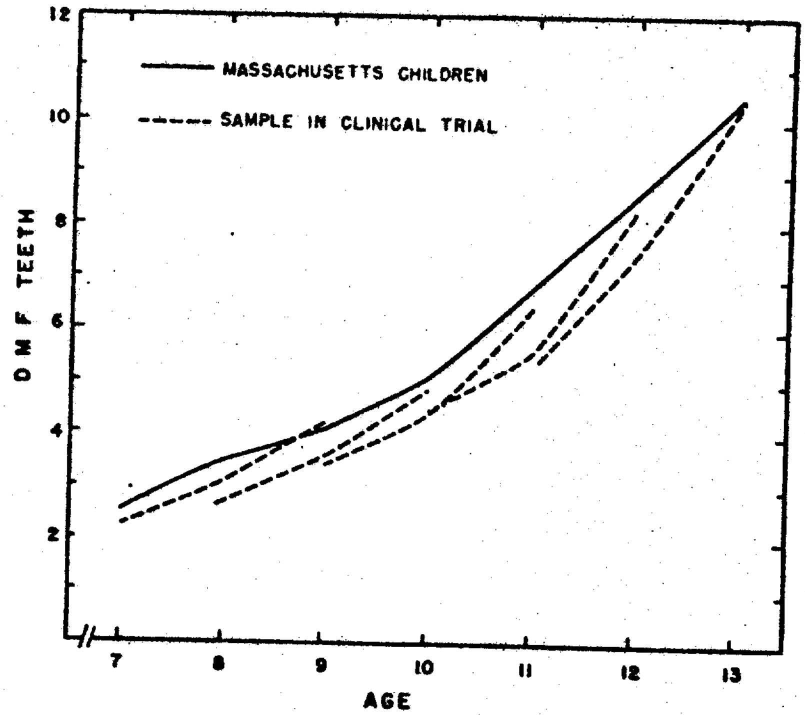

Fig 2 ▪ Mean counts of DMF teeth in Massachusetts children, 7 to 13 years of age, and sample participating in clinical trial. Solid line connects mean age-specific counts of DMF teeth for 8,934 Massachusetts children.[3] By age group, dashed lines connect mean age-specific counts of DMF teeth observed at three examinations carried out a year apart for 979 children in present study. For example, 7-year-old children were aged 8 at second examination and aged 9 at third examination.

Table 3 ▪ Number of boxes of cereal consumed per child per year, for 979 children.

	Regular	Presweetened	Total
First year			
Mean	16	18	34
SD	10	11	18
Minimum	1	0	6
Median	14	16	31
Maximum	115	90	206
Second year			
Mean	14	15	29
SD	10	11	18
Minimum	0	0	2
Median	12	13	26
Maximum	72	68	138

was slightly less than that of the presweetened cereals. However, a considerable range in estimated amounts consumed is evident for both regular and presweetened types. Cereal consumption generally was reduced somewhat during the second year. Each box of cereal on the average contained 10 oz of cereal, so numbers of boxes should be multiplied by 10 to determine intake in terms of ounces of cereal. More than 33,000 boxes were consumed by participating children during the first year and more than 28,000 boxes during the second year. The cumulative percentage distributions of children were plotted against amounts of cereal consumed (not shown); the shapes of the curves are similar for regular and presweetened cereals for each year.

Tables 4 and 5 show analyses of age, incremental caries, and needs for restorative treatment in permanent teeth and in permanent and deciduous teeth according to type and amount of cereal consumed. Low, medium, and high consumers are defined as previously specified.

During each of the two years of the study, the mean age of consumers in each category was nearly identical, differing only by no more than 0.1 of a year. Statistical analyses by analysis of variance resulted in F ratios less than the critical values, demonstrating that observed differences in age by group were nonsignificant.

Statistical analyses by analysis of variance of the two measures of incremental caries demonstrate no statistically significant differences among levels of incremental caries during the first or second year of the study. Examination of the rank order of the measures of caries increments during the two years encompassed by the study shows no consistent trend of caries incidence that suggests an association with cereal consumption.

Statistical analyses of the need for dental re-

Table 4 ■ Analyses of age, caries increments, and dental restorative treatment needs according to cereal consumption: first year.

	Low consumers		Medium consumers		High consumers		
	Mean	SD	Mean	SD	Mean	SD	F
Regular cereals							
Age	9.94	1.30	9.93	1.27	9.94	1.25	0.02
Net increment*	1.96	3.27	2.23	3.18	2.25	2.90	0.79
Caries incidence rate†	2.35	4.09	2.74	3.79	2.80	3.64	1.16
Permanent treatment	1.88	2.75	2.34	3.28	2.19	2.74	2.04
Total treatment	2.75	3.46	3.55	4.28	3.25	3.64	3.53
No. children	264		451		264		...
Range of consumption in boxes per year	1-9.2		9.3-19.1		19.2-115		...
Presweetened cereals							
Age	10.01	1.26	9.90	1.28	9.93	1.28	0.67
Net increment	2.01	3.89	2.15	2.80	2.34	2.82	0.73
Caries incidence rate	2.34	4.31	2.70	3.73	2.88	3.50	1.36
Permanent treatment	2.13	3.47	2.13	2.77	2.31	2.91	0.33
Total treatment	3.05	4.29	3.32	3.84	3.33	3.64	0.48
No. children	261		453		265		...
Range of consumption in boxes per year	0-9.8		9.9-21.7		21.8-90.0		...
All types of cereal							
Age	9.99	1.30	9.91	1.27	9.93	1.25	0.40
Net increment	2.02	3.83	2.05	2.80	2.50	2.87	2.12
Caries incidence rate	2.36	4.26	2.55	3.67	3.11	3.62	2.83
Permanent treatment	2.02	3.33	2.18	2.92	2.32	2.80	0.65
Total treatment	2.92	4.04	3.39	3.94	3.36	3.73	1.33
No. children	264		446		269		...
Range of consumption in boxes per year	6-20.7		20.8-39.8		39.9-206		...

*New decayed and filled surfaces.
†New decayed and filled surfaces per 100 surfaces at risk.

Table 5 ■ Analyses of age, caries increments, and dental restorative treatment needs according to cereal consumption: second year.

	Low consumers		Medium consumers		High consumers		
	Mean	SD	Mean	SD	Mean	SD	F
Regular cereals							
Age	10.91	1.31	10.92	1.26	10.99	1.25	0.32
Net increment*	4.24	4.87	3.84	4.16	3.53	3.93	1.79
Caries incidence rate†	4.30	4.54	3.88	3.86	3.55	3.92	2.28
Permanent treatment	4.38	5.42	3.81	4.72	3.52	4.07	2.23
Total treatment	5.06	5.67	4.36	4.88	4.12	4.30	2.65
No. children	263		451		265		...
Range of consumption in boxes per year	0-7.4		7.5-17.1		17.2-72.0		...
Presweetened cereals							
Age	11.01	1.28	10.91	1.30	10.91	1.21	0.64
Net increment	4.04	5.02	3.68	4.01	4.01	4.04	0.80
Caries incidence rate	3.91	4.56	3.81	3.90	4.07	3.86	0.34
Permanent treatment	4.48	5.95	3.73	4.38	3.57	3.98	2.90
Total treatment	4.98	5.99	4.35	4.64	4.21	4.33	1.89
No. children	263		452		264		...
Range of consumption in boxes per year	0-7.6		7.7-19.2		19.3-68.0		...
All types of cereals							
Age	10.94	1.30	10.95	1.28	10.90	1.24	0.14
Net increment	4.22	5.09	3.82	4.11	3.59	3.76	1.46
Caries incidence rate	4.14	4.61	3.92	3.94	3.65	3.73	0.98
Permanent treatment	4.33	5.81	3.89	4.52	3.45	3.94	2.27
Total treatment	4.90	5.92	4.46	4.74	4.12	4.27	1.68
No. children	263		446		270		...
Range of consumption in boxes per year	2.0-16.2		16.3-35.7		35.8-138.0		...

*New decayed and filled surfaces.
†New decayed and filled surfaces per 100 surfaces at risk.

storative treatment of permanent tooth surfaces demonstrate no statistically significant differences among groups with different levels of consumption. During the first year of the study, the difference among groups of regular cereal consumers in the need for restorative treatment of combined permanent and deciduous tooth surfaces is significant ($P = 0.05$). (According to statistical theory, 1 F ratio in 20 is expected to be significant because of chance alone.) However, no significant differences are observed among mean treatment needs of permanent and deciduous teeth according to grouping based on consumption levels of presweetened and all cereals. Examination of the rank orders of the mean levels of need for dental treatment in all categories of consumption during both years of the study reveals no consistent trend that suggests any association between caries incidence and cereal consumption.

Regression analyses of cereal consumption with the several measures of caries increments also showed no association between cereal consumption and caries incidence. The observed correlation coefficients were near zero, ranging from +0.08 to −0.07 and equally divided between positive and negative values. The correlations between age and cereal consumption were also near zero.

Discussion

As seen in Figure 2, the caries prevalence of the sample of participants is quite similar to that of the Massachusetts population. This similarity is remarkable in that the two sets of data are derived from two completely independent studies with two sets of examiners operating under their own diagnostic criteria. The data in Table 2 plus the plots in Figure 2 demonstrate that the participating children were susceptible to dental caries and that their caries prevalence was typical of that of children in a nonfluoride area.

As mentioned in the previous section, the cereal consumption of individual children was estimated from family size and orders of cereal. It is impossible to determine with accuracy the exact amount of food ingested except in metabolic ward studies where food intake is weighed. Although the procedure for estimating cereal intake in the present study may not provide exact measures of individual consumption, it does facilitate a rank ordering of participants according to level of consumption. This provides a means of categorizing children as low, medium, and high consumers. Interviews carried out by nutritionists verified this correlation between volume of cereal ordered and amounts consumed by the individuals.

Although some of the participating children consumed large amounts of cereals containing up to 45% sucrose, the results of the several analyses carried out demonstrate no association between dental caries and cereal consumption. During the two years, no significant differences nor any consistent pattern of differences in incremental caries were observed between children eating small amounts and those eating large amounts of cereal. This lack of differences is apparent in two measures of incremental caries, as well as in the needs for dental restorative treatment in permanent and deciduous teeth. No differences are observed in mean ages according to level of consumption; therefore, age is not a confounding factor.

These findings are not inconsistent with existing knowledge concerning caries etiology and findings of human dietary studies. Almost all of the studies on diet and caries have been carried out with experimental animals. The extrapolation of the findings to humans may or may not be appropriate because of significant differences in eating habits. Few long-term prospective studies have been reported in relation to diet and dental caries. The most comprehensive of these is the Vipeholm dental caries study,[5] carried out during five years in a Swedish mental institution. The authors of this classic study concluded that, although increased sugar consumption may increase caries activity, the risk is greater if the sugar is contained in a vehicle that tends to stick to the teeth and if the sugar is consumed between meals. They found that increased sugar consumption in solution at meals resulted in no increase in dental caries activity.

Finn and Jamison[6] concluded that a sugar-coated cereal does not produce a significant change in dental caries incidence when compared to noncoated cereals or fruits. The same conclusion of lack of demonstrable cariogenicity of cereals is drawn from the results of the present study. However, this conclusion must not be construed to dilute in any way the evidence associating dental caries with sucrose in general. Although some ready-to-eat cereals contain up to 45% sucrose, these cereals are eaten with milk 94% of the time.[7] The presence of milk may reduce the retentive characteristics of the cereals so that remaining food particles may be more rapidly cleared from the mouth. For the most part, cereals are eaten at mealtime. As shown in the Vipeholm study, sucrose taken at mealtimes and in other than a sticky vehicle failed to produce increased caries activity.

Many observational studies on diet and dental caries have been reported; these include findings of significant increases in caries experience in populations exposed to dietary changes involving increased consumption of highly refined carbohydrates.[8-10] However, the eating habits and the specific foodstuffs involved were not considered.

The results of the present study and those of other prospective dietary studies suggest that the cariogenicity of foods depends on factors other than sucrose content alone. The consistency of a food, the time of eating (at meals or between meals), plus accompanying foods (for example, milk) may somehow affect the cariogenicity expected as a result of sucrose content.

Certain dietary restrictions of highly refined carbohydrate intake may be extremely important in the control of rampant dental caries. However, it is impractical to eliminate all such foods from the diet, especially the diets of children. More extensive research in humans will be required to identify and to establish the rank order of the cariogenicity of different foods and eating habits. Such research is difficult, time-consuming, and expensive. This type of research is further complicated by the problems of obtaining approval from committees on studies involving human subjects, as there is a tendency to pass judgment on the cariogenicity of all highly refined carbohydrates before evidence from controlled experiments is available.

Conclusions

Under the conditions of the present study, no association was observed between dental caries incidence and the consumption of ready-to-eat cereals. In spite of the children's free access to unusually large quantities of regular and presweetened cereals, no increase in caries incidence was observed. The observed lack of cariogenicity of cereals may be due to their ingestion with milk and at mealtimes.

The cariogenicity of a foodstuff may not be highly correlated with the sucrose content alone. The time of eating, the consistency of the food, and the conditions under which a particular food is eaten may be as significant as its sucrose content in the determination of its relative cariogenicity.

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1. Peterson, J.K. North Dakota field test of cariostatic effect of 1% sodium dihydrogen phosphate and disodium hydrogen phosphate added to presweetened breakfast cereals. J Dent Res 48:1308 Nov-Dec 1969.

2. Glass, R.L.; Alman, J.E.; and Fleisch, S. The measurement of caries increments by counts and rates. Abstracted, IADR Program & Abstracts of Papers No. 633 March 1971.

3. Wellock, W.D. Dental Div, Massachusetts Dept of Public Health, House Doc no. 3902, 1967.

4. Kelley, T.L. The selection of upper and lower groups for the validation of test items. J Educ Psych 30:17 Jan 1939.

5. Gustafsson, B.E., and others. The Vipeholm dental caries study. The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. Acta Odontol Scand 11:232 Sept 1954.

6. Finn, S.B., and Jamison, H. The relative effect on dental caries of three food supplements to the diet. Abstracted, IADR Program & Abstracts of Papers No. 667 March 1969.

7. National Family Opinion. Cereal consumption study. Toledo, Ohio, National Family Opinion, 1972.

8. Toverud, G. The influence of war and post-war conditions on the teeth of Norwegian school children. II. Caries in the permanent teeth of children aged 7-8 and 12-13 years. Milbank Mem Fund Q 35:127 April 1957.

9. Holloway, P.J.; James, P.M.C.; and Slack, G.L. Dental disease in Tristan da Cunha. Br Dent J 115:19 July 2, 1963.

10. Harris, R. Biology of the children of Hopewood House, Bowral, Australia. 4. Observations on dental-caries experience extending over five years (1957-61). J Dent Res 42:1387 Nov-Dec 1963.

Dental Caries - Sugar

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Sugar and the Production of Bacteria in the Human Mouth

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Abstract. The aim of this study was to elucidate if sugar is a limiting substrate for the growth of the oral microflora. Six subjects were used. Water, glucose solution or peptone yeast extract broth were pumped into the mouth during 4-hour periods and all oral fluid was collected. The number of viable bacteria and the contents of RNA and DNA were estimated in the fluid. There was no obvious difference in the amount of microbial biomass shed into oral fluid during the water period and the glucose period, while an increased amount of biomass could be shown in the fluid during the broth period. Sugar does not appear to be the primary growth-limiting substrate of the oral microflora.

Key Words
Microbial ecology
Biomass production
Sugar

Introduction

The growth rate of bacteria is influenced by the concentration of the essential nutrients in the environment. MONOD [1949] has shown that the dependence of growth rate (μ) on substrate concentration (s) in an exponentially growing culture has the form of a Michaelis-Menten type function, i.e.

$$\mu = \mu_{max}\left(\frac{s}{K_s + s}\right)$$

where K_s is the saturation constant and μ_{max} is the value of μ, when s is no longer growth limiting. During the exponential growth phase the weight of bacteria formed is also directly related to the weight of substrate consumed [MONOD, 1949]. At steady-state growth in continuous culture there are similar relations between growth rate, yield of bacteria and growth-limiting substrate concentration [HERBERT *et al.*, 1956; TEMPEST, 1970].

When sugar is taken into the mouth, the oral microflora consumes the sugar and produces large amounts of acids [STEPHAN, 1940]. From this observation it has sometimes been implied that the growth rate and yield of bacteria in the mouth increases during sugar intake. However, this assumption takes for granted that sugar is the growth-limiting substrate in the mouth and, in fact, there is very little experimental evidence for that. The sugar may only be degraded in the glycolytic pathways without any growth of the microorganisms. Actually, HOTZ *et al.* [1972] have recently demonstrated that the concentration of sugar in pooled samples of dental plaque from 3,500 school children was so high, that sugar could not possibly be the limiting substrate for the growth of microorganisms in dental plaque.

The aim of the present study was to elucidate if sugar is a limiting substrate for the growth of the oral microflora, i.e. if sugar intake increases the production of bacteria in the mouth.

Materials and Methods

Collection of oral fluid. In addition to the two authors, four male second-year dental students were the subjects of this study. The subjects were instructed to thoroughly brush their teeth the evening before each experiment. They were not allowed to eat or drink or brush their teeth before the experiment started at 8 o'clock in the morning. Each subject got 0.8 g of paraffin (m.p. 52–54 °C), that was chewed for 5 min and the saliva produced was swallowed. Then a teflon tube (0.8 mm inner diameter) was introduced into the moth and the orifice of the tube placed in the maxillary sulcus above the second molar. Through this tube a test solution was pumped (Perpex pump, LKB-produkter AB, Bromma, Sweden) into the mouth at a rate of 27 ml/h. The subject's head was leaned forward and a silicone rubber tube (1.3 mm inner diameter) was held between the lips touching the front teeth. This tube was connected to a pump with a capacity of 105 ml/h. The subject was told not to swallow any fluid and chew the piece of paraffin to such an extent that fluid was continuously fed into the exit tube. This tube was led into a graduated test tube in an ice bath. As soon as 10 ml of fluid had been collected in the test tube, this was transferred into a cold bath (–35 °C) and then placed in a freezer (–20 °C). The second 10-ml sample collected and then a 10-ml sample was treated every half hour in a mixer (Whirlimixer, Fisons Scientific apparatus Ltd., Loughborough, UK) for 30 sec and 1 ml was used for determination of the number of viable bacteria in the sample. The rest of the sample was then immediately frozen as described above.

In three experiments, the following test solutions were used (1) water, (2) 1% (w/v) glucose solution, (3) peptone-yeast extract broth, containing per liter: 40 g trypticase (BBL, Cockeysville, Md.), 10 g yeast extract (Difco Laboratories, Detroit, Mich.), 5 g K_2HPO_4 and 10 g glucose.

In two subjects (2 and 6) also a 5-percent (w/v) sucrose solution was used in one experiment. All experiments were run for 4 h.

Extraction of nucleic acids from sediment of oral fluid. Every second sample of oral fluid was thawed in an ice bath and the samples from each hour of the experiments were pooled. To 40 ml of pooled sample from each hour 1 M perchloric acid was added to a final concentration of 0.2 M. The sample was centrifuged (20 min, 27,000 *g*, 4 °C) and the sediment was washed in 10 ml of cold (0 °C) 0.2 M perchloric acid. The nucleic acids in the sediment were extracted three times in 2 ml of 0.5 M perchloric acid at 70 °C according to the procedure recommended by HERBERT *et al.* [1971]. The extract was stored at −20 °C.

Determination of DNA. DNA in the perchloric acid extract of oral fluid was determined using the diphenylamine reagent as described by HERBERT *et al.* [1971] with 2-deoxy-D-ribose as standard.

Determination of RNA. The use of the orcinol reagent according to HERBERT *et al.* [1971] for determination of RNA in the perchloric acid extract of oral fluid showed that other sugars than ribose significantly intefered with this reagent. Therefore, the perchloric acid extracts of oral fluid were purified from most of the contaminating sugars before RNA was determined. To 1 ml of the extract, 65 mg of charcoal (No. 33033, BDH Chemicals Ltd., Dorset, England) in 1 ml of 0.5 M perchloric acid was added. The suspension was applied on glass wool in a Pasteur pipette and washed with 3 ml of water. The nucleic acids were eluted from the charcoal with 1.25 ml of a 50-percent (v/v) ethanol solution containing 0.1 N NH_4OH [EMERSON and HUMPHREYS, 1971]. For determination of RNA in the eluted material the orcinol and ferric chloride reagents of HATCHER and GOLDSTEIN [1969] were used. The reaction mixture was heated in boiling water for 20 min. D-Ribose was used as standard.

Determination of number of viable bacteria in oral fluid. 1 ml of the oral fluid was diluted in tenfold steps in 0.05 M potassium phosphate buffer (pH 7.0) containing 0.4% (w/v) sodium chloride. From each dilution, 10^{-5} and 10^{-6}, 0.1-ml samples were taken and cultured on the surface of three blood agar plates. The plates were incubated in a jar at 37 °C for 48 h under an atmosphere of 95% H_2 and 5% CO_2. The number of viable bacteria in 1 ml of oral fluid was calculated from the mean number of colonies growing on the plates.

Results

In most experiments saliva was produced to such an extent that almost a constant flow of oral fluid could be recovered from the subjects (table I). Only during the water period in subject 1 was the recovered amount of oral fluid significantly reduced (table I).

The experimental situation in each subject has to be considered as unique and the results are presented for each subject separately (fig. 1). Comparing the water period and glucose period no apparent difference could be established in the oral fluid when the number of viable bacteria

Table I. Amount of oral fluid recovered during the three experimental periods, in ml/h

Period	Subject 1				Subject 2				Subject 3			
	1 h	2 h	3 h	4 h	1 h	2 h	3 h	4 h	1 h	2 h	3 h	4 h
Water	60	57	59	64	95	98	97	96	85	87	83	87
Glucose	79	88	90	93	90	93	95	95	95	92	83	73
Broth	93	103	93	97	98	99	98	95	90	97	90	83
	Subject 4				Subject 5				Subject 6			
	1 h	2 h	3 h	4 h	1 h	2 h	3 h	4 h	1 h	2 h	3 h	4 h
Water	77	83	86	84	85	90	97	96	95	97	101	97
Glucose	95	94	91	83	95	95	92	90	98	102	104	101
Broth	92	92	89	87	95	90	93	100	93	100	99	98

as well as the contents of RNA and DNA in the fluid were estimated. During the broth period the number of viable bacteria increased in most subjects. Only in three subjects could an increase in DNA contents of oral fluid be shown during the broth period. In most experiments the change in RNA contents quite closely followed the change in the number of viable bacteria in the fluid.

In two subjects (2 and 6) also a 5-percent sucrose solution was used. The composition of oral fluid during the sucrose period was not significantly different from the composition of the fluid during the water period and glucose period.

Discussion

The yield of microbial biomass in a growing culture is directly related to the amount of growth-limiting substrate consumed [MONOD, 1949]. When a substrate is growth-limiting for the oral microflora, the addition of this substrate to the mouth should result in an increased production of microbial biomass. The produced biomass should be proportional to the amount of substrate added. However, no method exists for the measurement of the biomass of the mixed microbial flora inhabiting the mouth. This means that the production of microbial biomass cannot be directly assessed when a growth-limiting substrate has been added to the mouth. In the present study the production of microbial biomass in the mouth

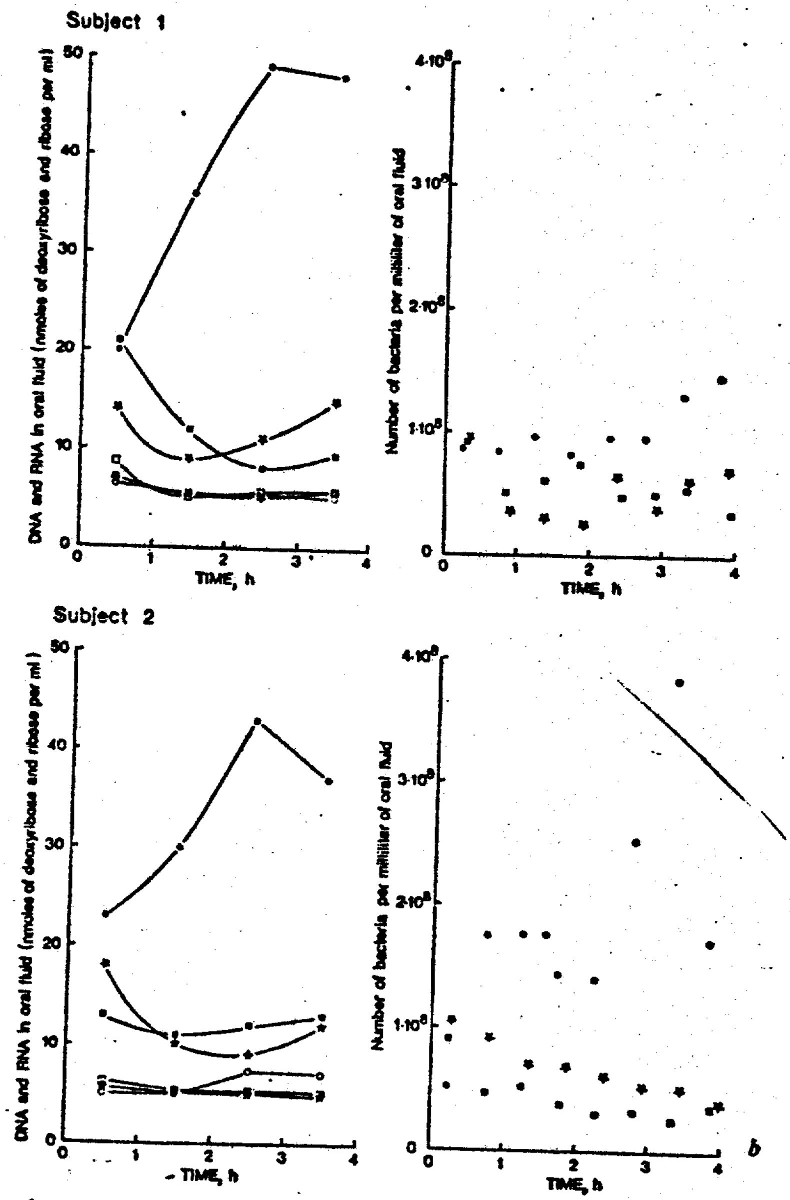
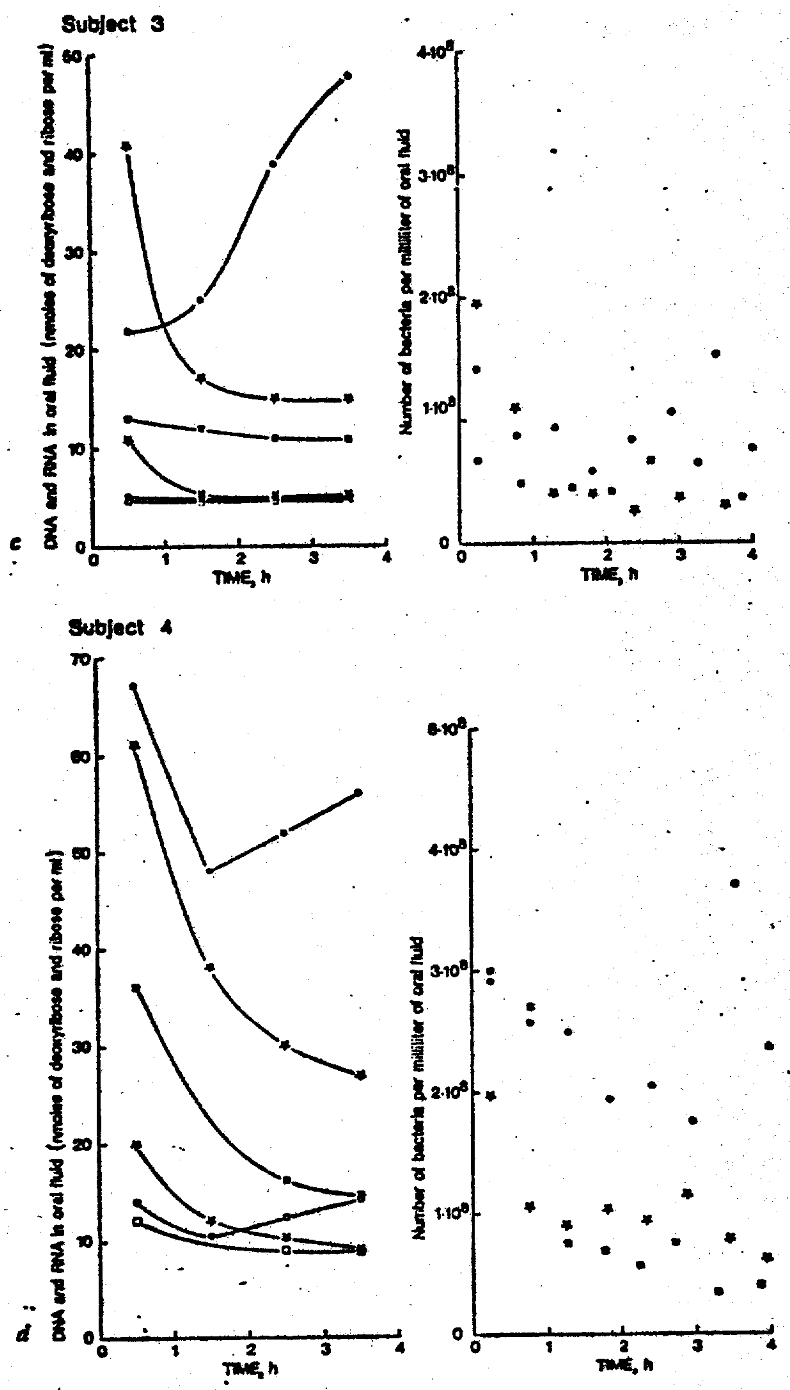


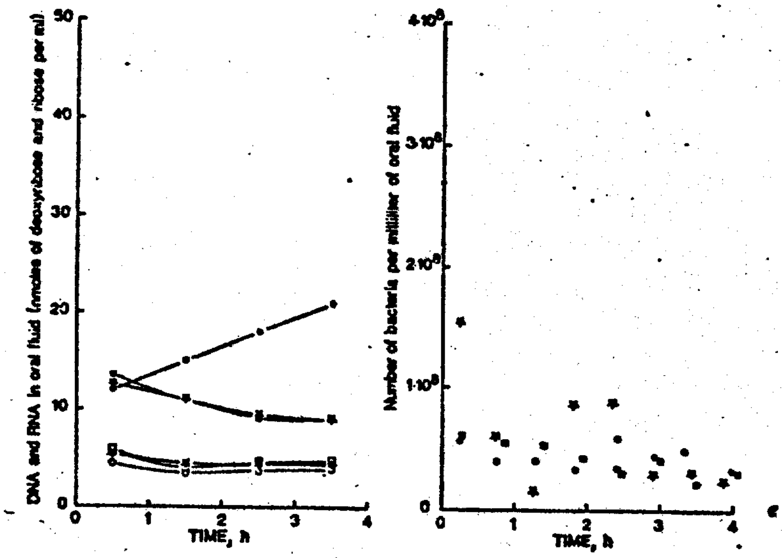
Fig. 1a–f. Composition of oral fluid in the six subjects; the contents of DNA (open marks) and RNA (full marks), as well as the number of viable bacteria during the broth (●), glucose (★) and water (■) periods.



Subject 3
c
DNA and RNA in oral fluid (nmoles of deoxyribose and ribose per ml)
TIME, h
Number of bacteria per milliliter of oral fluid
TIME, h
Subject 4
d
DNA and RNA in oral fluid (nmoles of deoxyribose and ribose per ml)
TIME, h
Number of bacteria per milliliter of oral fluid
TIME, h

Fig. 1 c–d

Subject 5



Subject 6

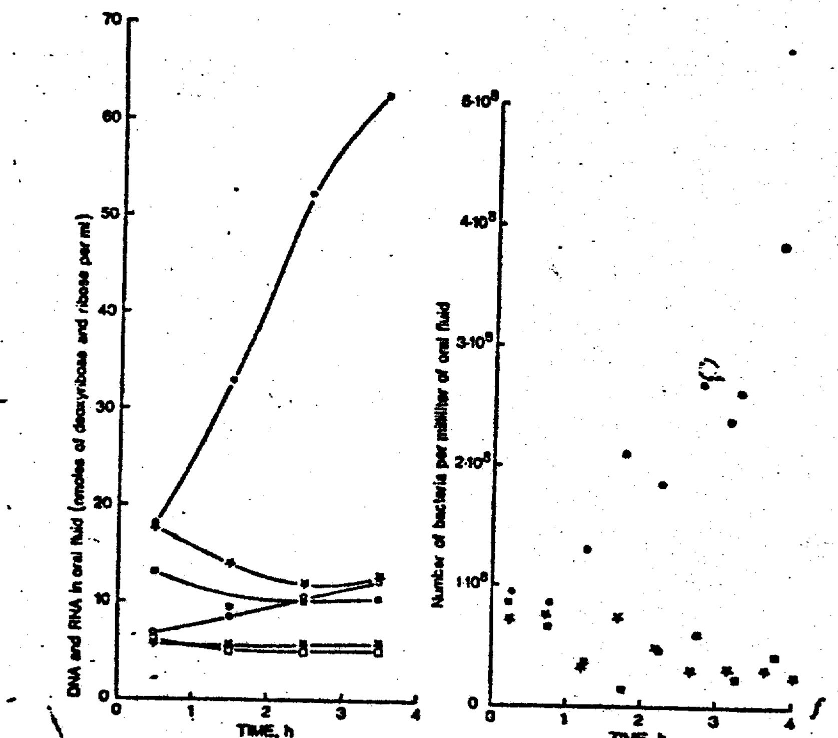


Fig. 1 e–f

during various nutritional conditions was estimated from the changes in the amount of microbial biomass shed into oral fluid during these conditions.

The dry weight of microorganisms most closely reflects the microbial biomass. However, the oral fluid contains nonmicrobial material such as glycoproteins and cell debris and this material is almost impossible to effectively separate from the microorganisms before a determination of the microbial dry weight is made. The bacteriological method to culture the microorganisms in oral fluid on artificial media only discloses the number of organisms in the fluid. The cell mass of the organisms on these media may be quite different from their mass in the actual habitat [SCHAECHTER *et al.*, 1958]. Furthermore, significant number of organisms in the oral fluid may not grow on the artificial media used. Chemical methods for evaluating the microbial biomass have the drawback, that there is no substance present in all organisms in an amount proportional to the amount of cell mass. The relative amount of protein as well as RNA and DNA in an organism changes with the growth rate of the organism [SCHAECHTER *et al.*, 1958]. The determination of these substances in the microorganisms of oral fluid is further complicated by the presence of nonmicrobial substances in the fluid.

The bacteriological and chemical methods used in the present study do not give a direct measure of the microbial biomass in the oral fluid. However, the combined figures on changes in RNA, DNA and number of viable bacteria during various nutritional conditions may warrant some conclusions. The concomitant increase of RNA, DNA and number of viable bacteria during the broth period must reflect an increase in the microbial biomass. In one subject, only an increase of RNA in the oral fluid could be demonstrated during the broth period. This could reflect an increase in biomass, but may as well be a charging of microorganisms with RNA before they increased their growth rates [KJELDGAARD *et al.*, 1958].

There was no obvious difference in the amount of microbial biomass shed into oral fluid during the water periods and glucose periods. This is in keeping with the finding that there is a high concentration of fermentable sugars in dental plaque [HOTZ *et al.*, 1972] and suggests that sugar is not the primary growth-limiting substrate in the mouth. The use of sucrose as test solution in two subjects confirmed this. The increased amount of dental plaque observed during sucrose intake [CARLSSON and EGELBERG, 1965] may be ascribed to the retention of bacteria to the tooth surfaces by extracellular polysaccharides produced from sucrose [GUGGEN-

HEIM, 1970]. However, there may exist ecological niches in the mouth where sugar is growth limiting. Such niches probably comprise a very small portion of oral microflora in the present subjects, but they may be more predominant in other individuals. However, there are reasons to believe that the food web or oral microflora is so complex that no single substrate is limiting for any significant portion of the oral microflora.

The concentration of bacteria in the oral fluid was surprisingly high at the end of the 4-hour water period. This indicates that nutrients in oral fluid are important for the maintenance of the oral microflora. To what extent nutrients in the diet may contribute to the maintenance of the flora is an open question. There are reasons to believe, however, that the effect of these nutrients may be quite marginal. The diet is usually kept in the mouth only for short periods [LANKE, 1957] and the nutrients available in the diet may not be the growth-limiting substrates which fit into the complex food web of the microflora.

Sugar in diet may influence the oral microflora in other ways than as growth-limiting substrate. Sugar may be consumed and acids produced without any microbial growth. Extracellular polysaccharides may be synthesized from sucrose and bacteria retained in the matrices formed. We think it is important that the various effects of sugar on the oral microflora are kept in mind when dietary measures are considered in dental caries prophylaxis.

Acknowledgements

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References

CARLSSON, J. and EGELBERG, J.: Effect of diet on early plaque formation in man. Odont. Rev. *16:* 112–125 (1965).

EMERSON, C. P. and HUMPHREYS, T.: A simple and sensitive method for quantitative measurement of cellular RNA synthesis. Analyt. Biochem. *40:* 254–266 (1971).

GUGGENHEIM, B.: Extracellular polysaccharides and microbial plaque. Int. dent. J., Lond. *20:* 657–678 (1970).

HATCHER, D. W. and GOLDSTEIN, G.: Improved methods for determination of RNA and DNA. Analyt. Biochem. *31:* 42–50 (1969).

HERBERT, D.; ELSWORTH, R., and TELLING, R. C.: The continuous culture of bacteria; a theoretical and experimental study. J. gen. Microbiol. *14:* 601–622 (1956).

HERBERT, D.; PHIPPS, P. J., and STRANGE, R. E.: Chemical analysis of microbial cells; in NORRIS and RIBBONS Methods in microbiology, vol. 5B, pp. 209–344 (Academic Press, London 1971).

HOTZ, P.; GUGGENHEIM, B., and SCHMID, R.: Carbohydrates in pooled dental plaque. Caries Res. *6:* 103–121 (1972).

KJELDGAARD, N. O.; MAALØE, O., and SCHAECHTER, M.: The transition between different physiological states during balanced growth of *Salmonella typhimurium*. J. gen. Microbiol. *19:* 607–616 (1958).

LANKE, L.: Influence on salivary sugar of certain properties of foodstuffs and individual oral conditions. Acta odont. scand. *15:* suppl. 23 (1957).

MONOD, J.: The growth of bacterial cultures. Annu. Rev. Microbiol. *3:* 371–393 (1949).

SCHAECHTER, M.; MAALØE, O., and KJELDGAARD, N. O.: Dependency on medium and temperature of cell size and chemical composition during balanced growth of *Salmonella typhimurium*. J. gen. Microbiol. *19:* 592–606 (1958).

STEPHAN, R. M.: Changes in hydrogen-ion concentration on tooth surface and in carious lesions. J. amer. dent. Ass. *27:* 718–723 (1940).

TEMPEST, D. W.: The continuous cultivation of microorganisms. I. Theory of the chemostat; in NORRIS and RIBBONS Methods in microbiology, vol. 2, pp. 259–276 (Academic Press, London 1970).

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Statistical Compilation to the Question of the Association Between Sugar Consumption and Dental Caries

Statistics show that the amount of sugar consumption is positively correlated with the incidence of dental caries. It is also known, however, that the many forms of sugar and the ways it is consumed have an extensive influence on caries generation, and that sucrose is not the only substance of importance in the etiology of dental caries. The following paper takes a new look at this problem in light of recent research results. The editors welcome the renewal in discussion on the topic as brought about by this paper and, thereby, the possible stimulation of research to further clarify questions pertinent to this issue.

The Editors

When considering the destruction of solid tooth tissue, enamel and dentin, sugar is said to be the most harmful of the carbohydrates. This is because the sugar that is broken down into different acids first loosens the homogeneous structure of the enamel crystal and eventually completely dissolves the hydroxyapatite. These may be the results of a few, goal-oriented experiments, but these experiments rarely represent the conditions actually existing in the mouth. Specific, in-vitro experiments will not be considered here; rather, I want to differ with the popular, statistically-based notions used to substantiate sugar's harmful effects on the teeth.

In addition to the afore mentioned experiments is the constantly reoccurring opinion (even today abundantly found in the literature) that the statistically signifigant decline of caries during the war was caused by food deficiency; in particular, to the great reduction in sugar consumption during that time.

The observation of a reduction in caries in Germany (1,2) during the second world war is joined by identical observations in ~~also~~ the USA (3), England (4), Sweden (5), Norway (6), Denmark (7) and Switzerland (8).

Most of the authors of these studies also associate reduced sugar consumption with the decrease of caries incidence. This theory is likewise advocated in the USA (L. J. Baume, 9).

On the other hand, there exists a group of serious researchers who, in light of their findings, are very skeptical or completely against this war-sugar-caries theory. Harndt (10, 11, 12) proved through his experiments on dogs, that caries fail to appear even when sugar is added to normal food in abnormal [excessive] amounts.

Similarly, Kantorowicz (13) and Spreter von Kreudenstein (14) categorically reject this theory.

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The main question to be clarified about this association is:

Was there really an ubiquitous decrease in sugar consumption during the war, especially there, were caries develope; that is, with each individual?

Here in Berlin, we are fortunate to have almost all the official documents of the former food officials relative to the past detailed rationing system. This offers to our study of the region of the former Reich's capital:

1) a representative population profile (the 1942 population being 4.5 million)

2) a relatively well defined area

3) a population that with few exceptions had to rely on only this food rationing system.

4) a city with a relatively well defined population fluctuation.

Food rationing was divided up for the following eight groups:

1) Children to 3 years of age

2) Children from 3-6

3) Children from 6-10

4) Children from 10-14

5) Youths from 14-18

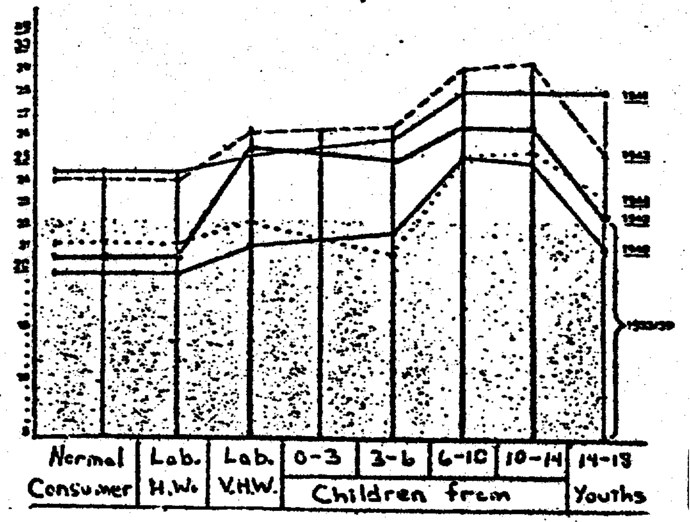
6) Normal consumers

7) Manual laborers - heavy work

8) Manual laborers - very heavy work

The largest group was that of the normal consumer. It was followed by groups 1-5 (the combined children and youths to age 18) and then the manual laborers - heavy work [group 7].

Figures from the pre-war years used for comparison are based on exactly registered import figures for the region of Berlin (see following table).



Sugar rationing among the different population groups in Berlin from 1939-1944. In comparison are the average figures for per-capita consumption of sugar from 1933-1939 (darkened areas).

The results show that the average amount of sugar allotted ranges from 23.06 kg/person (1944) to 25.65 kg/person (1941) a year.

Sugar imports into the city were from 21.29 kg (1933) to 22.86 kg (1939) per person a year. These figures also include industrially utilized sugar (bakeries and the sugar industry), so that the comparable per-capita consumption during pre-war times is much less than the consumption by sugar rationing between 1940 and 1944 (only entire war years are computed). The following should also be considered when considering nutrition during the war: Severely rationed fats, meat, and meat products were substituted with relatively large amounts of marmalade, beet syrup and artificial honey. These are sweets that, especially when taken together with sticky bread, have been viewed by various scholars as plainly constituting a caries-generating diet.

Marmalade rationing during the war was on the average 7.63 kg/person; during peace time, marmalade consumption is given at 3.6 kg/person a year. Therefore, during the war, consumption more than doubled.

The figures are even more divergent when considering artificial honey: 7.01 kg/person during the war, while being much less than 1 kg/person a year for the pre-war period, an almost meaningless amount.

According to these figures, one cannot conclude that a decrease in the consumption of sugar occurred in Berlin during the war. When, in addition, one computes the consumption of "substituted foods" - marmalade, beet syrup and artificial honey - the results even reveal a consumption increase of approx. 11 kg/person as compared with that of pre-war times.

In what ways does nutrition, in this case sugar, influence tooth decay, if at all?

1) by endogenous pathways; as a constitutional factor during development, especially during periods of mineralization. This applies to the mineralization of deciduous (baby) teeth, which begins with prenatal development and ends during the second year of life. Mineralization of permanent teeth begins at the time of birth and ends at about 12 years of age.

2) by exogenous pathways; as a local factor; namely, that foodstuffs, here sugar, or their metabolized products can directly affect teeth exposed in the mouth.

Regarding endogenous influences, it can be stated that according to our data, children and youths up to age 14 had almost normal nutrition and no shortage of sugar during the war years. The endogenous phase of the deciduous teeth coincides completely with wartime nutrition for those born in 1940 and is followed by a period of three years of the exogenous (local) phase. Those born in that year could only have had endogenous factors as influences on the tooth structure of their permanent teeth, as the 1. permanent molar first emerged in 1946.

Although according to our calculations sugar consumption was not less during war years than during peace time, 1951 statistics clearly show a distinct reduction in caries incidence beginning in 1941 and reaching its lowest point in 1946.

In conclusion the following can be said:

1) Many authors even today still directly associate the reduction of caries incidence during the war with an alleged decrease in sugar consumption.

2) The frequently expressed opinion that during the second world war a severe reduction in sugar use occurred, is not true, at least for the region of Berlin, the former Reich's capital. It can be assumed however, that the situation was similar in the other areas of Germany.

3) If one also includes the use of marmalade and artificial honey, the result shows a much greater consumption of "sweets" during the war than during the pre-war period 1933-1939.

4) The cause of caries reduction must therefore be sought in other realms; not, however, with the endogenous or exogenous effects of sugar.

by
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*This is difficult to reproduce. We have the original should FDA wish to borrow it.

Table 21.14

Decay experience and thumbsucking

Age until child sucked thumb	Proportion of five year olds with	
	No deciduous decay experience	Five or more deciduous teeth involved
Did not suck thumb	25% 688	42% 688
Under three years	27% 23	56% 23
Three years or more	[illegible]% 205	25% 205

The survey results relating to upbringing thus suggest that there are some habits which are associated with the disease experience of the child. Most of the factors shown to be related to disease were matters that were largely within the discretion of the mother, and although there was little suggestion that a single factor could eradicate the problem of decay it would seem that the mother could, if she realised and if she wished, choose to reduce the decay experience of her child.

21.4 Disease variation and current eating habits

In addition to the information that we obtained about early upbringing we asked a large range of questions related to the current eating habits of the child. It is unlikely that any individual variable is very important in relating eating habits to dental health, but by analysing these questions we hoped to find indicators of ranges of habits which tend either to help or hinder the cause of preventive dentistry.

One of the biggest problems in investigating the relationship between eating habits and dental health from survey data is that the total dental disease experience of the five year old has been affected by eating patterns since birth. If the child has already had dental problems, the mother may have been given advice which resulted in a change of eating habits. Such circumstances would then abrogate the expected relationship between eating habits and decay. In addition, of course, it is very difficult to obtain accurate information about eating habits since some mothers may give what they consider to be acceptable answers and mothers' views of what is large or small will vary; furthermore, the diversity of time, place and quantity of food consumed makes the collection of simple meaningful indicators very difficult. We feel in retrospect that for most of the meal questions, the information would have been better had we obtained a history of meals over a period of time and this was considered at the planning stage, but it would have distorted the balance of the interview to such an extent that the idea was discarded. If the main purpose of this inquiry had been to investigate variations in disease alone then eating habits would have justified much greater depth of study.

In terms of eating habits one of the factors of interest which emerged for the five year olds, was whether or not the mothers said the child usually had something to eat at break. About half of the children did. Among those who did not 32% had no decay experience and 34% had five or more teeth involved. Among those who did have something to eat at break 23% had no decay experience and 42% had five or more teeth involved with decay. Perhaps this is an indication that those who had something to eat at break were more used to having snacks between meals than the other children (see Table 21.15).

Table 21.15

Decay experience and eating at break

Whether child has anything to eat at break	Proportion of five year olds with			
	No deciduous decay experience		Five or more deciduous teeth involved	
Eats at break	23%	*443*	42%	*443*
Does not	32%	*470*	34%	*470*

We asked the mothers whether the child was used to eating a lot of cakes and biscuits, a fair number, or not many cakes and biscuits. Table 21.16 shows the relationship between this and the child's disease experience. Among the small group of children who were said to eat a lot of cakes and biscuits 21% of the children had no decay experience and 58% had five or more teeth involved. This compares with 30% decay free and 34% with five or more teeth involved among children who were said not to eat many cakes and biscuits. We also asked the mothers whether the child was allowed to help himself to biscuits or whether he had to ask first. Of the five year olds who were allowed to help themselves to biscuits only 18% had no decay experience and 50% had five or more deciduous teeth involved with decay. Thus the greater the quantity of and the more free the access to cakes and biscuits, the worse was the child's deciduous disease situation.

Table 21.16

Decay experience and consumption of cakes and biscuits

Consumption of cakes and biscuits	Proportion of five year olds with			
	No deciduous decay experience		Five or more deciduous teeth involved	
Eats a lot	21%	*46*	58%	*46*
Eats a fair number	24%	*317*	43%	*317*
Does not eat many	30%	*557*	34%	*557*
Helps himself to biscuits	18%	*170*	50%	*170*
Does not	30%	*751*	36%	*751*

Cakes and biscuits are not, of course, the only source of sweet things to eat, and we asked the mothers some similar questions about the child's fondness for sweets and how many he ate. The children said to be very fond of sweets were less likely to be decay free and more likely to have five or more teeth involved with decay than were the children who were said not to be as fond of sweets as most children (see Table 21.17). In terms of the amount consumed, the very small group of children who were said not to eat sweets at all were at a very noticeable advantage; 48% of them were decay free and only 14% had five or more teeth involved with decay. The children who were said to eat only a small quantity of sweets were also at an advantage. The children who were said to eat a large quantity of sweets were at a considerable disadvantage, 42% having five or more deciduous teeth involved with decay.

Table 21.17
Decay experience and consumption of sweets

Consumption of sweets	Proportion of five year olds with			
	No deciduous decay experience		Five or more deciduous teeth involved	
Very fond of sweets	22%	302	42%	302
About average	30%	525	38%	525
Less fond than average	31%	92	31%	92
Eats a large quantity	26%	62	42%	62
Eats a medium quantity	22%	209	38%	209
Eats a small quantity	27%	513	40%	513
Eats a very small quantity	39%	102	33%	102
Does not eat sweets	48%	21	14%	21

We have used these few illustrations of particular facets of eating to indicate that there are relationships between eating habits and dental health and that they can be shown from survey data. As illustrations we would not wish them to be interpreted too narrowly. We feel that the relationship between eating habits and dental health is based on much wider factors than sweets, biscuits and cakes and that the survey results are merely indicative that there are certain dietary factors, which are within the control of the mother, that do affect dental health.

In Chapter 10 we showed that mothers were aware that eating sweet things was associated with decay. They also showed that their reaction to stopping decay was not to restrict the intake of the sweet foods but to clean the teeth. We feel it would be appropriate to end this section by examining whether or not the children who were said to brush their teeth most frequently were at an advantage with respect to total deciduous decay experience.

Table 21.18
Decay experience and frequency of toothbrushing

Frequency of toothbrushing	Proportion of five year olds with			
	No deciduous decay experience		Five or more deciduous teeth involved	
Three times a day	28%	44	36%	44
Twice a day	28%	469	37%	469
Once a day	27%	297	42%	297
Less than once a day	29%	98	35%	98

Table 21.18 shows that there was no systematic variation in disease experience with variation in toothbrushing frequency.

22 Summary of findings and conclusions

Chapter 1

Introduction

The survey findings are based on the results of dental examinations conducted on a random sample of 13,000 children aged 5-15 in maintained schools in England and Wales, and on an interview carried out with 3,000 of the mothers of children aged five, eight, twelve and fourteen. Seventy dentists were seconded from their duties in the School Dental Service to carry out the dental examinations and they all attended a one week training course to practice using the definitions laid down for the survey examination so as to achieve as uniform a standard of examination as possible.

The response achieved for the survey was very high, 95% of the children who were selected for examination were examined, and for 91% of those selected for both parts of the inquiry we obtained both the dental examination and the interview with the mother.

22.1 Summary of findings

Chapter 2

Dental development

The survey included children ranging from the age of five to fifteen, thus covering the whole period of transition from deciduous teeth to permanent teeth. The rate at which this change takes place varies very much from child to child. The first deciduous tooth to exfoliate naturally is usually one of the lower central incisors, a few of these teeth were already missing among children who were only just five years old, on the other hand, a few lower central incisors were still present among children who had recently passed their seventh birthday.

The eruption of permanent teeth occurs at different ages for different children. In general it appeared that for boys the eruption of permanent teeth tended to occur a little later than for girls.

Chapter 3

Dental decay and treatment

By far the greatest amount of contact between children and the dental profession arises because of tooth decay. At the time of the survey about two-thirds of children were found to have some active decay, the proportion being lowest at 57% among the fifteen year olds and highest at 78% among the eight year olds. Among the five to eight year olds more than 20% of children had five or more actively decayed teeth, among children aged nine to fifteen more than 10% had five or more actively decayed teeth.

The treatment available for decayed teeth is either restoration by filling or extraction. The proportion of children who had some filled teeth increased over the age groups from 26% of five year olds to 88% of fifteen year olds. The proportion of children with no filled teeth at the age of fourteen and fifteen was 19% and 12% respectively. The figure for the fourteen year olds is probably the best estimate of the proportion of children who are obtaining no restorative treatment since the fifteen year olds who are still at school do not represent the total age group because the early school leavers are excluded.

The survey only measured the extraction experience for permanent teeth since it is impossible to tell for children in the process of changing from one dentition to the other whether the missing deciduous teeth were exfoliated or extracted. Among the oldest children, the fourteen and fifteen year olds, about a third had already had some permanent teeth extracted, and at an age as early as seven, eight and nine years old some permanent teeth were being lost.

Teeth in different positions in the mouth contribute very differently to the decay experience of the child and this is so for both deciduous and permanent teeth. In both dentitions it is the molars which are most disease prone.

Chapter 4

Total decay experience

If one takes as an indication of total decay experience the sum of current disease and the evidence of past treatment then one can see that very few children avoid decay entirely. Seven out of ten five olds already have some evidence of decay experience, at the age of eight nine out of ten children have evidence of decay experience and among the teenagers fewer than five children in a hundred have no evidence of decay experience.

As well as knowing how many children have had some decay it is of interest to know the extent of their disease experience. For the permanent dentition this can be achieved by summing the number of teeth with current decay or evidence of past treatment but for the deciduous dentition the examination provided no estimate of the number of deciduous teeth that had been extracted for decay reasons. For the five year olds, among whom little natural exfoliation would have so far taken place we estimated the likelihood of the missing deciduous teeth being diseased. This provided an estimate of the proportion of five year olds who had ten or more teeth involved with decay, a level often used to indicate rampant decay in the deciduous dentition. By this method we estimate that 11% of five year olds had ten or more deciduous teeth, that is a half or more of the deciduous dentition, involved with decay.

Chapter 5

Accidental damage

In some cases children need dental attention because of damage caused to the teeth by some kind of accident. It is most likely that the front teeth are the ones to suffer in this way and the dental examiners found that about one in ten girls and one in five boys among the twelve to fourteen year olds had evidence of such damage. This relatively high involvement with accidental damage is influenced by the fact that the criteria for the dental examination included fractures of the enamel which are fairly minor kinds of damage and would probably not require treatment and would perhaps be hardly noticeable.

Chapter 6

Dentures

Among all the children examined for the survey, that is about 13,000, only 46 children were found to have dentures; of these 23 had apparently suffered accidental damage to the teeth, 5 had developmental or congenital problems and 18 needed the denture because of extractions due to decay. Only one child had a full upper denture the others all had a partial denture involving one jaw.

Chapter 7

The condition of the gums

Another of the problems that affect dental health is the condition of the gums. The examiners recorded whether the child had any gum inflammation, debris or calculus. It is possible that the survey dental criteria for recording gum conditions were more stringent than those which are currently applied in practice. From the age of seven onwards about threequarters of children were involved with gum trouble of one or other kind. Gum inflammation and debris were found to be significantly associated.

Chapter 8

Orthodontics

Not all children have the good fortune to have trouble free dental development and the most common condition that arises is that of teeth crowded together. As many as 65% of eight year olds were recorded as having some crowding, and over the age range seven to fifteen the proportion was always greater than a half. Taking all the orthodontic assessments into account the dental examiners were asked to say whether, in their opinion, the child needed (or would need) orthodontic treatment. The future orthodontic need of five and six year olds was, of course, rather difficult to assess but by the age of seven about a half of the children were estimated to be in need of treatment. The proportion was highest among eight year olds where 57% were said to be in need of treatment. The need for orthodontic treatment may be apparent some considerable while before treatment is actually carried out and so the proportion in need of such treatment decreased among the older children till it reached 28% and 27% of fourteen and fifteen year olds. By that age any treatment that was going to be carried out would most likely have been started and so about a quarter of the children are likely to remain with an orthodontic treatment need that is unmet. A quarter of fourteen year olds had previously had some orthodontic treatment compared to the fact that 28% were said to be currently in need of treatment, thus about half of the estimated total orthodontic need is being met. Chapter 17 shows that mothers did not always recognise the need for orthodontic treatment which the dentists had found.

Chapter 9 *Overall dental condition*

It was of interest to assess what the total current need for dental attention was at the time of the survey and for this estimate we included the three most common sources of dental need, that is active decay, some gum trouble, or some orthodontic treatment need. By combining these three grounds for dental need we found that eight out of ten five year olds and nine out of ten children in the age range six to fifteen would benefit from dental treatment or oral hygiene instruction.

Chapter 10 *Mother's dental experience, knowledge and attitudes*

Nearly all children have their first permanent front teeth before the age of eight, many have them at the age of five or six. Similarly nearly all children have their first permanent back teeth by the age of eight and again many have them at the age of five or six. We asked the children's mothers when they thought these teeth erupted and over three-quarters said the first front teeth erupt before the age of eight but under a quarter estimated that the first permanent back teeth come through as early as that. There would thus appear to be a considerable lack of knowledge among mothers about their children's dental development. Mothers who were themselves good dental attenders were no more likely than other mothers to be aware of the pattern of permanent tooth eruption, neither were mothers in the top social class group more likely than others to know these facts. We felt that in many cases the mothers either did not know that any back teeth erupted at the age of six, or mistook them for the last of the deciduous teeth. In fact among twelve year olds who had already had some of their back permanent teeth extracted 45% of the mothers stated that all the child's extractions so far had been deciduous teeth.

In terms of knowledge about decay mothers were in no doubt that it could occur in very young children. Half of the mothers thought that such trouble could start before the age of three and three-quarters thought decay could start before the age of four. Neither were the mothers in much doubt as to what caused decay, eight out of ten blamed eating sweets and sweet things. When asked what might be done to help prevent decay the majority suggested better tooth cleaning, the minority suggested restricting the intake of sweet things.

Chapter 11 *Mother's views on the dental care of her child*

We asked the mothers whether they had any preference for different kinds of treatment for their children. Over a half said that if a bad back deciduous tooth was involved they would prefer it to be extracted. Only about one in ten said they would prefer extraction if the tooth concerned was a bad permanent tooth. In view of their different attitudes for the different dentitions it is particularly ironic that mothers are not aware which teeth are which.

The preference for different kinds of treatment reflected the mother's own attendance pattern to a considerable extent, for example a half of the mothers who were themselves regular attenders preferred fillings for deciduous teeth whereas only a quarter of mothers who were irregular attenders preferred that. Once permanent teeth were involved, eight out of ten of even those mothers who themselves had the worst dental attendance pattern said they would prefer their child's bad back permanent teeth to be filled.

We asked the mothers whether they thought that their children currently needed any dental treatment, six out of ten mothers said the child did not need any treatment. Only about four out of ten children had in fact been classified by the dental examination as currently free from decay.

Chapter 12

The child's dental background

Over the age of five very few children have not seen a dentist whether for the relief of pain or for conservation. In fact the proportion who were said to have never been to the dentist was 29% among the five year olds, 9% among the eight year olds, and 3% among the twelve and fourteen year olds. Beyond the age of five it may be that the more accurate description is that the child had seldom been to the dentist since some of the children who were said never to have been to the dentist had in fact had some fillings and extractions.

We asked the mothers whether the children had ever, in the past, had any extractions either of deciduous or permanent teeth. A quarter of five year olds, two-thirds of eight year olds and three-quarters of twelve and fourteen year olds had at some time had at least one extraction. Having teeth extracted thus soon ceases to be a minority experience. Even the children of mothers who were themselves regular attenders had not noticeably escaped the experience.

The two main services through which children can obtain dental treatment are the General Dental Service and the School Dental Service, although, in fact, the majority of treatment is carried out through the General Dental Service. Mothers (or children) can decide to change the service they use whenever they so wish. Three-quarters of children had at some stage used the General Dental Service and the proportion who had at some time used the School Dental Service reached 48% among the fourteen year olds. Among fourteen year olds a half of the children had always used the General Dental Service and just under a quarter had always used the School Dental Service and just over a quarter had used both.

Children of mothers who were themselves regular attenders were much more likely to have always used the General Dental Service, as were children in the top social class group and children in London and the South East. Conversely children who had always used the School Dental Service were more likely to come from less dentally aware backgrounds. When asked the reason for using the type of dental service the child attended the major reason for going to the General Dental Service was that the mother took the child to her own dentist. The reasons for going to the School Dental Service were that the mother was notified of the need to see a dentist by the school, that the school dentist was more convenient or that the school dentist was thought to be good with children.

Chapter 13

Visiting the dentist

Among those children who had been to the dentist nearly two-thirds said that the reason for the most recent visit was for a check-up. Between a fifth and a quarter of the different age groups said their last visit had been because of dental trouble and about one in ten were prompted to go because of a note from school. As might be expected the note from school was of more importance as a stimulus for dental attendance among children who are in the habit of using the School Dental Service. These figures reflect the fact that the majority of treatment is obtained through the General Dental Service, and also some of the differences in the organisation of the two services.

The treatment that was said to have been received as a result of the most recent dental visit was highly associated with the reason for the visit. For children whose last visit was said to have been for a check-up fewer than ten per cent had treatment which involved extractions whereas among those whose last visit was prompted by dental trouble over a half had some extractions.

Chapter 14

Toothbrushing

When asked about prevention of decay many mothers put considerable faith in toothbrushing. We asked the mothers how frequently the children brushed their teeth and about a half said twice a day and about a third said once a day. The survey results showed no marked variation between the frequency of toothbrushing and decay, but there were significant differences in relation to gum inflammation and debris.

Chapter 15

Toothache

We asked the mothers whether the children had ever had toothache and for a half of them the answer was in the negative. Over a third of the children whose mothers were regular attenders had had toothache which suggests that toothache does not always arise in circumstances of dental neglect on the part of the parent. In two-thirds of all cases the outcome for the tooth when a child had toothache was extraction.

Chapter 16

Mother's awareness of accidental damage

We asked mothers whether the children had ever suffered any accidental damage and compared their answers with the examination findings. Among children said by the dentist to have traumatised incisors approximately half of the mothers said there had been no accidental damage. We would have expected that the mothers would have been aware of any serious accident and so we scrutinised the cases where there was disagreement between the dentist and the mother and found that in the majority of cases the level of trauma recorded by the dentist was very minor and could fairly easily have escaped the notice of the mother.

Chapter 17 *Mother's views on orthodontics*

Chapter 5 has shown the level of orthodontic need as assessed by the dental examiners and this chapter discusses the mothers' attitudes to orthodontics. Mothers apparently attach considerable value to the treatment of children's teeth if they are crooked or protruding. They did not, however, have the same level of assessment as the dentists as to which conditions required orthodontic treatment. Among children aged twelve and fourteen who had previously had no orthodontic treatment but whom the dentist considered needed some, half of the mothers said the child had no orthodontic irregularity. Since the dentist's assessment for orthodontic need could arise from several sources we looked at one particular orthodontic assessment and examined whether the mother and dentist agreed about the child's teeth being crowded, but even where the dentist said there was crowding in the upper middle segment, that is among upper canines and incisors, nearly a half of the mothers said there was no irregularity.

Chapter 18 *Dental background and dental health*

Among the five year olds a considerable proportion of children had not as yet been to the dentist, but among the eight, twelve and fourteen year olds about half the children were said to have been for a check-up within the past six months. There was, on the other hand, a considerable proportion of children in the older groups who were not going to the dentist unless they had some trouble or were prompted by a note from school. This was so for about a third of children aged eight, twelve and fourteen. Among the fourteen year olds, 12% were said not to have been to the dentist at all within the previous two years.

In terms of the proportion of children who would benefit from some dental attention, that is those with some decay, some gum trouble or some orthodontic treatment need, the level was very high even among the potential regular attenders. This was also the case for the gum trouble need and the orthodontic need when examined on their own. In terms of the proportion of children with some active decay the disparity between attendance patterns was greater but the proportion of children with active decay among the potential regular attenders was disappointingly high. For fourteen year olds a half of the regular attenders had some active decay, as had two-thirds of the occasional attenders and three-quarters of the irregular attenders.

It is fairly common to find that if one calculates disease experience by adding current decay to evidence of past treatment then the most dentally well cared for children contain the highest proportion of children with extensive disease experience. This arises when the stage of decay at which some fillings are provided is earlier than the level used to detect current decay. For example, 32% of fourteen year olds who had been for a check-up in the last six months had ten or more teeth with decay experience compared to 22% of the occasional attenders and 25% of the irregular attenders.

We have already seen that the mothers' attitudes towards preferences for dental treatment vary according to whether deciduous or permanent teeth are involved. Among eight year olds 66% of potentially regular attenders had some decayed deciduous teeth, 35% having some deciduous teeth that were unrestorable. Among the eight year olds who were potential irregular attenders 76% had some decayed deciduous teeth, 43% having some that were unrestorable. The differences between the attendance patterns were thus relatively small.

The proportion of children with fillings in deciduous teeth varied between attendance patterns especially for the five year olds, being 43% for those who had been for a check-up in the last six months, 11% for those whose check-up had been longer ago than that and 18% for those who were not in the habit of going for a check-up. By the age of eight the differences were less marked being 57%, 41% and 33% for the attendance types respectively. The interesting fact here is that even among the children who have been for check-ups the proportion with deciduous filled teeth is not much greater than a half.

In terms of the filling experience of permanent teeth the advantage that the potentially regular attenders have over the children with other attendance patterns is to be seen among fourteen year olds where the regular attenders have, on average, 6.1 filled permanent teeth compared with an average of 3.3 filled permanent teeth among the irregular attenders. For the fourteen year olds who have not been to a dentist in the past two years the average number of filled teeth is as low as 1.0.

The alternative to the restoration of decayed teeth is the eventual loss of them by extraction. The proportion of children who have had some permanent teeth extracted for decay reasons by the age of fourteen varies with attendance pattern. However, even among the regular attenders a quarter have lost some permanent teeth by that age. For the irregular attenders, the proportion having lost some permanent teeth by the age of fourteen was as high as 40%.

Chapter 19 — *Regional variations in the dental condition of children.*

Dental attendance patterns among children were found to vary between the regions. London and the South East had a higher proportion of potential regular attenders and Wales had proportionately fewer. Conversely, Wales had considerably more irregular attenders proportionately, for example among the fourteen year olds in Wales 40% were irregular attenders compared to 22% in London and the South East.

The examination results revealed that the proportion of children with some active decay, some filled teeth and some extracted permanent teeth also varied with region, children in London and the South East being in the most advantageous position and those in Wales being in the least. For example, the proportion of fourteen year olds with some extracted permanent teeth was 50% in Wales but 18% in London and the South East.

The variation in treatment experience between the regions was not entirely accounted for by the uneven distribition of attendance pattern. In general, regular attenders had fairly similar treatment experience wherever they lived but irregular attenders were more likely to have less current decay and more restorative experience if they lived in London and the South East. For example the proportion of children aged fourteen who had some filled permanent teeth taking account of the three attendance patterns was 90%, 83% and 78% in London and the South East compared to 83%, 73% and 50% in Wales. In this respect the North, Midlands and East Anglia and Wales and the South West tended to be similar to Wales.

One factor for which regional variation affected the regular attenders as well as the irregulars was the proportion of children who had lost some permanent teeth. For fourteen year olds in the three main attendance groups the proportion who had had some permanent teeth extracted was 18%, 11% and 28% in London and the South East, but 40%, 43% and 63% in Wales.

Chapter 20

The dental condition of individual tooth types

When the individual tooth types are examined to see which ones are most prone to disease there is no doubt that the first permanent molars bear the brunt of both disease and treatment experience among children. It is interesting to find that there is little regional variation in the total disease experience of the first permanent molars in different regions but considerable evidence of treatment variation. Children in London and the South East retain many more of their first permanent molars, fewer of which are currently decayed, many more of which are filled, than is the case in other regions.

We looked particularly closely at the dental condition of fourteen year olds since they were the last full age group in school at the time of the survey and thus represent the best estimate we have of the dental condition of adolescents as they approach adult life. An examination of the dental condition of different tooth types for fourteen year olds of different attendance patterns from different regions revealed that those who were well cared for appeared to be more decay prone because of the variation in the level of decay at which fillings take place. The disease experienced by children who do not go for dental check-ups was more likely to be untreated than was the case for children who go for regular check-ups.

In view of the history of the first permanent molar it was of interest to investigate the condition of the tooth posterior to it, the second permanent molar. Among regular attenders aged fourteen over half of the second permanent molars already had evidence of disease experience, the great majority of which had been treated restoratively. Among irregular attenders of the same age there was less evidence of disease but, except in London and the South East, the majority of disease had not been treated restoratively. Only in a small proportion of cases had extractions of second permanent molars occurred, the majority of diseased teeth being currently decayed.

At the time that the survey dental examination was carried out the majority of decayed teeth found among fourteen year old irregular attenders were still restorable, so although the fourteen year olds who are not accustomed to going to the dentist for a check-up had considerably more current treatment need than the regular attenders the position was not for the most part irretrievable.

Chapter 21

Disease variation among five year olds

A detailed analysis was carried out to investigate what factors were associated with the estimated total deciduous disease experience of five year olds. It was found that social and background factors such as the mother's own dental attendance pattern, the parents' occupation and educational attainment were associated with disease experience. It was also found that disease experience was associated with region. The analysis that was carried out to see whether factors other than social and background were associated with the regional variation in disease was rather inconclusive.

Different methods of upbringing and various current eating habits showed that some patterns of behaviour were more associated with disease than others. For example, if the child had been in the habit of having drinks from a bottle beyond the age of two the likelihood was that the level of disease experience would be high. On the other hand if the child had been a thumb sucker beyond the age of three the likelihood of disease was fairly low. If the child ate something at school break-time the disease experience was higher than if he did not. If the five year old ate few or no sweets the likelihood of decay experience was lower, whereas if the child helped himself to biscuits the disease experience was higher.

None of the indicators are intended to be interpreted in isolation but they have been used to illustrate the fact that exposure to certain conditions has certain results. The results which we show may well not be direct cause and effect relationships, for example, the fact that a child of five is free to help himself to biscuits may merely reflect that he has had the opportunity to develop a liking for sweet food which could well be manifested in many different ways. Merely stopping him from helping himself to biscuits may not improve the situation.

22.2 Conclusions

One of the main purposes of the children's survey was to provide information about the dental health of children which would complement that obtained from the survey among adults, thereby showing at what stage the characteristics which so markedly distinguish adults of different attendance patterns in different regions are observable among children.

Many of the dental attitudes that people have are transmitted from parents to children very early in life. Even by the age of five many decisions have been made as to the child's dental attendance pattern and attitudes towards treatment. Some changes of attitude do take place during the child's school life and the survey results show that some children with not very good dental attendance patterns are receiving restorative treatment; but the gap between the dental condition of those who attend regularly and those who do not is apparent even among the five year olds, among whom 43% of children who have recently been for a check-up have some filled deciduous teeth compared to 18% of the children who do not go for a check-up. Although all the deciduous teeth are eventually lost the recollection of their dental treatment and the resulting expectations of dentistry remain. As the permanent dentition develops one finds that the children with the best dental background have more restorative treatment and less current decay but no less disease. However, by the age of fourteen the variation in dental condition between children of different attendance patterns and different regions has not for the most part reached irreparable dimensions (except for the state of the first permanent molars). In theory the vast majority of children at this age could be made dentally fit and have a reasonable expectation of adequate reliance on natural teeth well into adult life, provided that resources were available to carry out the backlog of treatment needed and the children were prepared to have the treatment.

However in the real world resources are limited and children's dental attitudes have already been moulded by their experiences and expectations. The fact that at the age of fourteen the situation would still appear to be retrievable for most children does not mean that the future situation will in fact be saved. In the survey of adult dental health carried out in 1968 the differences between the treatment received by young people aged 16-34 according to their attendance pattern and the region in which they lived were more marked than among the fourteen year olds in 1973. The young adults had a wider range of permanent teeth extracted and a wider range of teeth extensively filled. It is not possible to say how far the fourteen year olds will in the next few years recreate that pattern, it is only possible to say that at fourteen the position was still redeemable. It is hard to say how far the attitudes of the fourteen year olds are already entrenched in terms of the priorities attached to dental welfare but it is worrying to compare the regional variation in loss of permanent teeth among fourteen year olds with the regional variation in total tooth loss among adults.

Table 22.1
Tooth loss among fourteen year olds and total tooth loss among adults, by region

Region	Proportion of fourteen year olds with some permanent teeth extracted in 1973		Proportion of adults edentulous in 1968	
The North	37%	*308*	46%	*864*
Midlands and East Anglia	38%	*207*	34%	*629*
Wales and the South West	34%	*145*	43%	*431*
London and the South East	18%	*263*	28%	*1008*

The regional variations that have come to light from the analysis of the survey results were known to exist before the survey was carried out, for the survey

Table 22.2
Regional variation in treatment carried out through the General Dental Service

Region		Ratio of conserved* to extracted deciduous teeth for children aged 0-4		Ratio of conserved* to extracted deciduous teeth for children aged 5-15		Ratio of filled* to extracted permanent teeth for children aged 5-15	
North	The North	1.12		0.55		5.09	
North West		1.47	1.43	0.88	0.78	5.72	5.91
Yorks & Humberside		1.56		0.79		6.95	
West Midlands	Midlands and East Anglia	1.95		1.06		6.87	
East Midlands		2.06	2.16	1.06	1.12	7.38	7.21
East Anglia		3.62		1.51		8.05	
Wales	Wales and the South West	1.58	2.53	0.94	1.54	4.25	6.30
South West		3.44		2.11		8.49	
G.L.C.	London and the South East	10.70	7.04	3.40	2.82	17.04	13.40
South East		5.55		2.47		11.38	
Wales	Wales	1.58	1.58	0.94	0.94	4.25	4.25

* *Treatment carried out under the General Dental Service as reported in the Dental Estimates Board Annual Report for 1972*

findings reflect the same variations as are shown by the ratios of restoration to extraction in the dental treatment carried out within the General Dental Service and reported in the Dental Estimates Board Annual Report 1972, and indeed in previous annual reports. However the context in which to interpret them was not known until background information and the overall levels of disease among children were established. Since the survey results show no massive variation in disease experience regionally which could have accounted for the regional variation in the ratios these variations must be due to treatment rather than disease.

If one compares the regional variation in treatment with the regional variation in population per dentist and pupils per school dentist then maybe one should not be surprised to find that in the south conservative treatment has been provided for a much larger and more varied group of children than is the case in other regions.

Table 22.3
Regional distribution of manpower resources

Region		Persons per GDS* dentist		Pupils per school dentist†	
North	The North	5783	5395	5633	6189
North West		5210		6260	
Yorkshire and Humberside		5414		6539	
West Midlands	Midlands and East Anglia	5602	5578	6653	6633
East Midlands		5765		7315	
East Anglia		5161		5485	
Wales	Wales and the South West	5736	4437	5093	4552
South West		3828		4201	
G.L.C.	London and the South East	2682	3159	4829	5207
South East		3717		5511	
Wales	Wales	5736	5736	5093	5093

* *General Dental Service*
† *Pupils at 1st January 1973, whole time dentist equivalents at 31st December 1972*

The practising dentist sees in a clinical setting the dental situation that we have described from the survey results. He is making his patients reasonably dentally fit or relieving pain in situations of emergency. Since even among the children who are regular attenders we found that nearly half had some current decay it is not surprising that the dentist's time is fully taken up with the treatment requirements of the children already on his books.

Without the information which a survey can provide it is very difficult to tell what proportion of children are repeatedly dropping through the protective net of restorative dentistry. Among fourteen year olds 12% were said not to have seen a dentist in the previous two years and their restorative experience was very limited compared to other groups of children. It is, on the other hand, encouraging to see that among the 21% of fourteen year olds who have been to the dentist fairly recently although prompted by dental trouble or a note, eight out of ten have some filled teeth and are therefore receiving care and attention despite their attitudes and backgrounds.

Many of the policies which have attempted to tackle the problems of scarce resources have advocated limiting restorative dentistry to those children who demonstrate favourable dental attitudes and behaviour. Any assessment of priorities on this basis accentuates the marked division in dental attitudes thus perpetuating the limited expectations that some sections of the community have of dental health. The problem for the future, which is by no means a new one, is how to reduce disease and how to increase treatment resources in order to provide the full amount of restorative treatment shown to be needed among children.

Epidemiologic Study of Dental Caries Experience and Between-Meal Eating Patterns

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The relationship between dental caries and between-meal snacks was investigated in a study of 1,486 high school students. The participants completed a questionnaire on between-meal habits and then were given dental examinations. The lack of differences in dental caries between racial and geographic groups was not related to the frequency of sucrose-containing, between-meal snacks.

Dental caries has been designated as a bacterial disease, although there is no agreement as to the specific etiologic organisms involved.[1] As a result of recent research, information on the mechanism of the activity of caries and the organisms concerned has been obtained. Bacteria, such as streptococcus strains, have been found to cause lesions on the smooth surfaces of animals' teeth; bacteria are dependent only on sucrose for the production of dextrans.[2,3] Dextran binds bacteria together and causes them to stick to the teeth; sucrose is the principal substrate for the production of acids that attack and destroy the substance of the teeth.[4] Consistent with these findings is the fact that the progress of lesions in populations is related directly to the presence and frequent use of sticky sugars in the diet.[5] Prevalence of caries has been associated with the amount and frequency of high sucrose-containing, between-meal snacks.[6] Caries experience is associated with between-meal eating so that differences or lack of differences between racial and geographic groups might be related to the amount and frequency of sucrose-containing between-meal snacks. The purpose of this study was to test the hypothesis that dental caries is related to the consumption of sucrose-containing between-meal snacks.

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Materials and Methods

A total of 1,486 white and black high school students in Detroit, Michigan and Columbia, South Carolina who were 14 to 17 years of age completed a questionnaire on between-meal eating habits. They then were examined for dental caries.

Dental examinations were conducted with the aid of a portable chair and a dental spotlight; radiographs were not used. Caries experience was recorded by use of the Klein and Palmer code.[7] Only obvious lesions in which soft dentin could be detected were recorded as carious. An assistant recorded the dental findings.

The drinking waters of both cities' contained no fluoride until about one year before the survey. To obviate the effect of ingestion of fluoride on the prevalence of caries, only residents who had consistently used the city's water since birth were included in the sample. The effect of outside sources of fluoride was controlled by the information on residency and permission slips and by questioning the participants. Some participants were excluded as a result of incomplete histories or because they had used water other than the city's.

Total caries experience, as indicated by DMF teeth, may not be related closely to the consumption of sucrose, because all lesions that are found in the mouth are included. This problem was anticipated in the early stages of the study, and therefore, dental data also were recorded and coded according to smooth surface and proximal lesions. The DMF teeth of each person and

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the mean DMF teeth of each group were calculated according to age, sex, race, and geographic location.

Information about the amount and frequency of consumed sucrose-containing between-meal snacks and cariogenic foodstuffs was gathered by use of a self-administered questionnaire. The questionnaire was easily understood and it was filled out as indicated during previous pretests. Standardized conditions were used to ensure uniformity before a dental examination was given.

The main purpose of the questionnaire was to determine snacking patterns in the form of between-meal eating habits. The students were asked to indicate if food items, such as candy, cake, ice cream, bread, crackers, and so forth were never eaten, sometimes eaten, or usually eaten between meals. The study design was intended to determine a general pattern, rather than attempt to acquire information that concerned specific amounts of consumed food. The prevalence of caries experience was compared with differences in between-meal patterns of eating, particularly the frequency and types of sucrose-containing foods.

Results

Findings for the analysis of DMF teeth are indicated in Table 1 and have been discussed in a previous report.[8] In Detroit, significant differences were found for race and age, but there was no significant difference between the sexes. No significant differences were found in data from Columbia. When taken as a whole, the mean DMF teeth for Columbia was slightly higher than the mean value for Detroit.

The results from the questionnaire about between-meal eating were examined to determine if a difference or a lack of difference found in caries experience could be associated with the frequency and amount of high sucrose snacks.

Several preliminary analyses were conducted to determine if patterns of response followed specific trends. A review of several hundred questionnaires resulted in a decision to classify responses according to the presence or absence of sucrose. Specific items were categorized as sticky sugar, sugar, and low sugar. The results of a preliminary analysis are shown in Table 2. This classifi-

TABLE 1
MEAN NUMBERS OF DMF TEETH

Age Groups	White			Black		
	No.	Mean	SD*	No.	Mean	SD
Males in Detroit						
14	30	11.13	5.19	55	8.56	4.00
15	39	10.56	5.10	47	8.85	3.71
16	18	10.39	4.37	32	10.22	4.69
17	21	11.33	2.82	16	10.19	3.76
All ages	108	10.85	...	150	9.45	...
Males in Columbia						
14	32	10.81	4.31	61	10.34	4.20
15	61	9.23	3.76	64	11.37	3.90
16	36	12.28	4.27	42	11.10	3.61
17	29	10.79	4.25	30	11.47	5.35
All ages	158	10.78	...	197	11.07	...
Females in Detroit						
14	27	10.15	3.33	92	8.61	4.10
15	38	11.39	3.75	105	8.74	3.97
16	25	12.00	3.91	57	11.11	5.40
17	18	12.83	4.62	24	10.67	4.85
All ages	108	11.59	...	278	9.78	...
Females in Columbia						
14	32	11.16	3.59	74	11.07	3.98
15	55	11.13	5.43	103	10.71	4.28
16	73	11.08	3.98	76	11.61	4.67
17	31	10.23	4.36	43	11.84	4.39
All ages	191	10.89	...	296	11.20	...

* SD, standard deviation.

TABLE 2
SNACKS CONSUMED BY 15-YEAR-OLD BLACK FEMALES FROM COLUMBIA

Favorite Snack	Classification
Candy	Sticky sugar
Cookies	Sticky sugar
Cake	Sticky sugar
Soda pop	Sugar
Ice cream	Sugar
Gum	Sugar
Chocolate milk	Sugar
Potato chips	Low sugar
Apples	Low sugar
Hot dogs	Low sugar
Hamburgers	Low sugar
Milk	Low sugar
Cheeseburgers	Low sugar
Sandwiches	Low sugar
Fish sandwich	Low sugar
Orange juice	Low sugar
Grapes	Low sugar
Crackers	Low sugar

Note: Total number of individuals who responded was 100.

cation corresponded to the hypothesis that persons with a high caries experience would have consumed more sugars and sticky sugars than persons with a low caries experience.

Because the plotted frequencies of DMF teeth showed approximately normal distributions, data could be separated into low, medium, and high caries experience according to the mean and a half standard deviation above and below the mean. Those scores that were greater than a half standard deviation were designated as high caries experience, those a half less were designated as low, and those within a half standard deviation were designated as medium. The dental data were separated initially by age, sex, race, and location. Because the data were separated, ages, sexes, races, and locations could be combined for further analysis based on low, medium, and high caries experience; data separation also minimized the effect of longer exposure of the teeth of older age groups to carious attack.

Chi-squares were calculated to determine if the differences in total caries experience (DMF teeth) were associated with snacking habits, as elicited by 25 items on the questionnaire for white and black males and females in Detroit and Columbia. Two hundred chi-squares were calculated. Although 14 of the chi-squares indicated significant differences, only 6 of these were significant with regard to the pattern of the hypothesis, ie, that caries experience would be higher for groups that reported more frequent use of sucrose-containing snack items.

The next step in the analysis was to examine the relationship between the responses from the questionnaire and the number of low, medium, and high proximal lesions.

Because of this non-normal data, the usual statistical calculation of the mean and standard deviations could not be used. The group with no lesions was designated as low caries activity. The remainder of the individuals were combined, a mean was calculated, and then the group was separated into medium and high caries activity according to this mean. On this basis, the computer was programmed to indicate the relationship of the responses from the questionnaire to low, medium, and high caries activity by age, sex, race, and location. These individual groupings then were combined by age, then by sex, then by race, and finally by location.

Chi-squares were calculated to test for significant relationships of responses on between-meal eating patterns that were indicated in the questionnaire to low, medium, and high proximal caries experience. These calculations were done for anterior proximal, posterior proximal, and anterior and posterior proximal caries experience. Tests were done according to sex and race for both Detroit and Columbia.

Chi-squares were calculated to determine if differences in proximal caries experience were associated with snacking habits, as elicited by 25 items on the questionnaire for white and black, males and females in Detroit and Columbia. Six hundred chi-squares were calculated. Although 61 of the chi-squares indicated significant differences, only 36 of these were significant with a regard to the hypothesis, ie, that caries experience would be higher for groups that reported more frequent use of sucrose-containing snack items.

Chi-squares then were calculated to determine if differences or lack of differences in caries experience between whites and blacks were related to snacking habits, as elicited by 25 items on the questionnaire. Entire groups of whites were compared with entire groups of blacks in Detroit and Columbia. Fifty chi-squares were calculated. Most of these analyses indicated that there were

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Milk
Tea

significant differences between whites and blacks in snacking patterns as reported in the questionnaire. Caries experience in Detroit was somewhat higher for whites than blacks (11.22 DMF teeth vs 9.31 DMF teeth). Therefore, it would be expected that more white than black children consumed sucrose items. Among the 25 chi-squares calculated for Detroit, 16 were statistically significant. Only six of these significant chi-squares, however, indicated that the pattern of response was consistent with the hypothesis that blacks would consume less sugar-containing and more nonsugar-containing items (Table 3).

Unlike Detroit, caries experience in Columbia was similar for whites and blacks (10.74 DMF teeth vs 11.14 DMF teeth). Chi-squares would be expected to show little difference in snacking patterns between whites and blacks.

But, 24 of the 25 chi-squares that were calculated for Columbia indicated considerable differences in snacking habits between white and black children. In 14 of the significant chi-squares, black children reported that they consumed more sucrose items and less nonsucrose items than white children (Table 4).

Discussion

Studies with isolated populations, such as Fisher's report[9] on Tristan da Cunha, have demonstrated that as a population changes its way of life, which includes dietary habits and sharp increases in the consumption of

TABLE 3
SIGNIFICANT DIFFERENCES FAVORING OR OPPOSING HYPOTHESIS THAT CONSUMPTION OF SUCROSE BETWEEN MEALS IS ASSOCIATED WITH A HIGHER CARIES EXPERIENCE IN WHITE CHILDREN (Detroit)

Significant Chi-square Values				
Favor Hypothesis		Do Not Favor Hypothesis		
Item	P Value	Item	P Value	Nonsignificant Chi-square
Usual snack	<0.001	Spending money	<0.001	No. of meals eaten
Breakfast cereal	<0.001	Between-meal frequency	<0.01	Snack like to eat
Sweet potatoes	<0.001	Favorite snack	<0.10	Pop
Potato chips	<0.001	Candy	<0.001	Peanut butter sandwich
Crackers	<0.05	Cake	<0.001	Fresh fruit
Bread	<0.05	Gum	<0.001	Milk
		Jelly	<0.001	Tea
		Cookies	<0.001	Other snacks
		Pie	<0.001	Food eaten every day
		Ice cream	<0.001	

TABLE 4
SIGNIFICANT DIFFERENCES IN REPORTED BETWEEN-MEAL SNACKING OF WHITE AND BLACK CHILDREN IN COLUMBIA

Significant Chi-Square Values				
Whites Favoring More Sucrose		Blacks Favoring More Sucrose		Nonsignificant Chi-Square, Neither Group Favoring Sucrose
Item	P Value	Item	P Value	
Usual snack	<0.001	Spending money	<0.001	Food eaten every day
Pop	<0.02	Number of meals eaten	<0.001	
Peanut butter sandwich	<0.05	Snack like to eat	<0.001	
Breakfast cereal	<0.001	Between-meal frequency	<0.001	
Sweet potatoes	<0.001	Favorite snack	<0.001	
Potato chips	<0.001	Candy	<0.001	
Bread	<0.001	Cake	<0.001	
Fresh fruit	<0.001	Gum	<0.001	
Milk	<0.01	Jelly	<0.10	
Tea	<0.001	Cookies	<0.001	
		Crackers	<0.02	
		Pie	<0.001	
		Ice cream	<0.001	
		Other snacks	<0.02	

sucrose, there is a noticeable increase in caries. It is plausible that the blacks in this study have changed their way of life from previous generations and that they have adopted more of a white man's diet; this would include a significant increase in the consumption of sucrose between meals. With an increase in industrialization and urbanization, blacks have become more assimilated into an urban way of life than in past generations. In this setting, sucrose-containing foods become more attractive and available for consumption.

Socioeconomic status does not appear to have been an influencing factor in the results of this study, because all samples were selected from schools that are located in the lower economic areas of the cities. Schools that are located in the upper or middle socioeconomic areas were not used for the survey. Although a few participants with a higher socioeconomic status have been included because of residence, the total sample was considered to be of low socioeconomic status. Even though the socioeconomic status of the samples that were studied may not be identical, the populations generally were in the lower socioeconomic status in their respective cities. Studies have indicated that socioeconomic status has little or no effect on the total DMF teeth, but it is related to differences among the components of the DMF count; decayed and missing teeth scores are higher for low socioeconomic groups and filled rather than decayed teeth scores are higher for upper socioeconomic groups.[10,11]

Other investigators have concluded that the relationship of sucrose to dextran that is formed in the presence of oral streptococci facilitates the carious process by causing bacterial plaque to stick to smooth surfaces of enamel.[4,12,13] Cariogenic streptococci were thought to be responsible for the initiation of smooth surface caries, but they will only produce dextran in the presence of sucrose.[1,3,12,14] Sucrose consumption, therefore, might be associated more closely with caries in smooth surfaces alone than with caries scores that include lesions in pits and fissures, in which other bacteria are thought to play an etiologic role. Because of this finding, it seemed that consumption of sucrose would be related more closely to carious activity on smooth surfaces than to lesions in pits and fissures. Chi-square results, however, did not support this hypothesis.

Specific cariogenic streptococci must be present, in addition to high intakes of sucrose, in the oral cavity to facilitate the initiation of caries on smooth surfaces.[4,15-17] It can be hypothesized that increases in caries that were found among blacks in this study were a result of infection with specific bacteria that were responsible for dental caries in the presence of high and continuous intakes of sucrose.

Conclusions

This study was designed to obtain information about between-meal snacking patterns and to relate the differences or lack of differences in caries experience between groups with the consumption of sucrose between meals. To examine this relationship between sucrose and dental caries experience, the between-meal questionnaire was developed and administered to acquire information about the snacking habits of the populations studied.

The results from the analyses of questionnaire responses, in relation to caries experience, were not as expected. No significant relationship could be found between the consumption of sucrose-containing between-meal snacks and low, medium, and high caries experience. Analyses were conducted for total caries experience as expressed in DMF teeth and also for proximal (smooth surface) caries experience, which was thought to be related more closely to the consumption of sucrose. No clear pattern of association was found in any of these analyses. Only a few of the chi-squares that were calculated were significant; this would be expected only as a result of chance. The caries experience that was recorded in this study was the result of a lifetime exposure to the oral environment. Changes in oral flora and dietary patterns or habits, however, could occur in a short period of time. Past caries experience, therefore, does not indicate necessarily a strong relationship to snacking habits, which could have changed recently.

An examination of the data according to race, however, indicated that whites and blacks consumed a great number of between-meal snacks. Blacks in Detroit and Columbia consumed more snacks of all kinds (sucrose and nonsucrose) than whites. The results from chi-square tests revealed that an equal number of significant values supported and negated the hypothesis that high caries experience would be related to a higher consumption of sucrose snacks between meals.

In this study, caries experience was not associated with between-meal eating patterns. Lack of differences between racial and geographic groups was not related to the frequency of sucrose-containing between-meal snacks.

References

1. FITZGERALD, R.J., and JORDAN, H.V.: Polysaccharide-Producing Bacteria and Caries, in HARRIS, R.S. (ed): *Art and Science of Dental Caries Research*, New York: Academic Press, 1968, pp 79-86.
2. DONAHUE, J.J.; KESTENBAUM, R.C.; and KING, W.J.: The Utilization of Sugar by Selected Strains of Oral Streptococci, abstracted, IADR *Program and Abstracts of Papers*, No. 58, 1966.
3. GIBBONS, R.J., and BANGHART, S.: Cariogenicity of a Human Levan Forming Streptococcus and a Streptococcus Isolated from Subacute Bacterial Endocarditis, abstracted, IADR *Program and Abstracts of Papers*, No. 137, 1967.
4. CARLSSON, J.: Plaque Formation and Streptococcal Colonization on Teeth, *Odontol Revy* 19:1-14, 1968.
5. GUSTAFSSON, B.E., et al: The Vipeholm Dental Caries Study. The Effects of Different Levels of Carbohydrate Intake on Caries Activity in 436 Individuals Observed for Five Years, *Acta Odontol Scand* 11: 232-264, 1954.
6. WEISS, R.L., and TRITHART, A.H.: Between Meal Eating Habits and Dental Caries Experience in Preschool Children, *Am J Public Health* 50:1097-1104, 1960.
7. KLEIN, H., and PALMER, C.E.: Studies on Dental Caries: X. Procedures for Recording and Statistical Processing of Dental Examination Findings, *J Dent Res* 19:243-256, 1940.
8. BAGRAMIAN, R.A., and RUSSELL, A.L.: An Epidemiologic Study of Dental Caries in Race and Geographic Area, *J Dent Res* 50: 1553-1556, 1971.
9. FISHER, F.J.: A Field Survey of Dental Caries, Periodontal Disease and Enamel Defects in Tristan da Cunha, *Br Dent J* 125:447-453, 1968.
10. KELLY, J.E.; VAN KIRK, L.E.; and GARST, C.C.: *Decayed, Missing, and Filled Teeth in Adults, United States—1960-1962*, USPHS (National Center for Health Statistics, Series 11, No. 23), Washington, DC: Government Printing Office, 1967.
11. KLEIN, H., and PALMER, C.E.: Community Economic Status and the Dental Problems of School Children, *Public Health Rep* 55: 187-205, 1940.
12. JORDAN, H.V., and KEYES, P.H.: In Vitro Methods for the Study of Plaque Formation and Carious Lesions, *Arch Oral Biol* 11: 739-801, 1966.
13. STEPHAN, R.M., and HARRIS, R.M.: Location of Experimental Caries on Different Tooth Surfaces in the Norway Rat, in SOGNNAES, R.F. (ed): *Advances in Experimental Caries Research* (A Symposium Presented on December 29, 1953 at the Boston Meeting of the AAAS), Washington, 1955, pp 47-65.
14. FROSTELL, G.; KEYES, P.H.; and LARSON, R.H.: Effect of Various Sugars and Sugar Substitutes on Dental Caries in Hamsters and Rats, *J Nutr* 93:65-76, 1967.
15. CARLSSON, J.: Effect of Diet on Presence of Streptococcus Salivarius in Dental Plaque and Saliva, *Odontol Revy* 16:336-347, 1965.
16. WINTER, G.B.: Sucrose and Cariogenesis, a Review, *Br Dent J* 124:407-411, 1968.
17. WOOD, J.M., and CRITCHLEY, P.L.: The Extracellular Polysaccharide Produced from Sucrose by a Cariogenic Streptococcus, *Arch Oral Biol* 11:1039-1042, 1966.

A survey of the relationship between caries prevalence
and the consumption of sugar and other foods by man
has been interpreted to show that snack foods share importance
with sucrose in caries causation.
Support for this conclusion is found in animal experiments
and some in vitro and in vivo tests.

The cariogenicity of snack foods and confections

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Since the time of Pierre Fauchard, observant dentists have concluded that caries activity and patients' diets are related. The validity of this conclusion has been established by research workers who have shown that changes in diet produce noticeable increases in caries in human or animal populations. In spite of agreement on the importance of food habits in caries causation, the specific mechanisms by which foods activate caries or the characteristics of foods that make them more or less harmful to teeth are still uncertain. The problem for dentists who want to give their patients up-to-date, practical dietary advice on caries prevention is complicated by the introduction of a greater variety of new foods in recent decades than in any other comparable period. In an effort to determine whether today's patterns of food use indicate the need for changes of emphasis in diet counseling, I have brought together pertinent findings from epidemiologic and diet studies in man, from animal tests, and from in vitro and in vivo experiments.

Epidemiologic and diet studies

Caries occurred before there were any refined carbohydrates. It has been found in wild animals and in the hominids of the Pleistocene era,[1] and it affected the majority of the pre-Columbian Indians in the region that is now western New York.[2]

The suggestion that sucrose had special importance in caries causation was made by Fauchard and other early dentists who noted that caries was most common in the mouths of the wealthy who alone could afford to buy much sugar. The belief that sucrose was the prime cause of dental decay was strengthened by increased caries occurrence during the 18th and 19th centuries as the sugar trade developed with the New World.[1] However, other changes also were taking place in the diet besides the increased consumption of sugar; potatoes and refined wheat flours were coming into more common use.

Further emphasis on the role of sucrose in caries causation resulted from observation on the year-to-year changes in caries incidence that occurred in European countries before, during, and after the First and Second World Wars[3,4] when the supply of sugar was sharply limited. Although caries figures fell pretty much in parallel with sugar consumption, a clear case cannot be made because, as Toverud[4] has pointed out, between-meal eating was virtually eliminated, and, in addition, refined flour was removed from the diet. Either of these alone might have reduced the strength of the caries attack.

The relation of wartime sugar rationing to caries also has been studied in Japan.[5] In the year that the sugar intake was lowest, at about 5 ounces a year, figures about new caries in first molars were lowest and increased proportionately as the sugar allowance rose to its maximum of about 30 lb a year. As in other studies of this sort, the effect on caries of the elimination of between-meal eating or reduction in the use of other potentially cariogenic foods cannot be ignored.

More comprehensive information on time changes in caries incidence in relation to alternatives of food patterns cannot be found. Although the federal services or professional associations are not able to provide information on year-to-year changes on caries prevalence, reliable information on the production and consumption of different foods can be obtained from the US Department of Commerce and the US Department of Agriculture. Thus, it is possible to cast some light on food relationships to caries by correlation of such incomplete information as is available on time or geographic differences in caries activity with reliable information on food use.

Some diverse information indicates that the caries attack rate is increasingly active where there is no fluorine in the drinking water. Dental examinations of freshmen entering the University of Minnesota[6] (Fig 1) showed about 20% more DMF teeth in 1939 than in 1929 and also between 1949 and 1939. The increase was smaller between 1949 and 1959, when it would be expected that increased exposure to fluorine was bringing about a reduction of caries. J. K. Peterson, according to a letter of June 1972, has reported increases in caries in North Dakota during the past 25 years that include a 50% increase between 1933 and 1953 in Barnes and Walsh counties and an increase in DMFT in 15-year-olds elsewhere from 7 to 11.5 between 1940 and 1959. The finding by Glass and co-workers[7] of no change in caries prevalence over 25 years, in spite of the use of fluorine in dental offices, by prescription, or in dentifrices, also can be interpreted as evidence of an increase in the strength of the caries attack. In Connecticut, Potgieter and co-workers[8] found an increase in caries in 11- to 16-year-old children between 1944 and 1950.

If there has been such an enhancement of the caries attack, what has happened on the sugar side of the equation? Surprisingly, none of the several governmental or trade reports shows any significant change in per capita sugar consumption during the past 50 years[9] (Fig 2). Something besides total sugar intake must be playing an important part. I suggest that this unknown factor is the way sugar is now being used and the associated increase in types of manufactured foods or snack foods that have come into common use.

Two interrelated facts support that explanation. One is that today, unlike in earlier years, the bulk of sugar goes into manufactured foods instead of being used in the home. Figure 3 shows that the manufacturer's share has risen from 25% in 1910 to almost 70% today.[9] Sugar is used

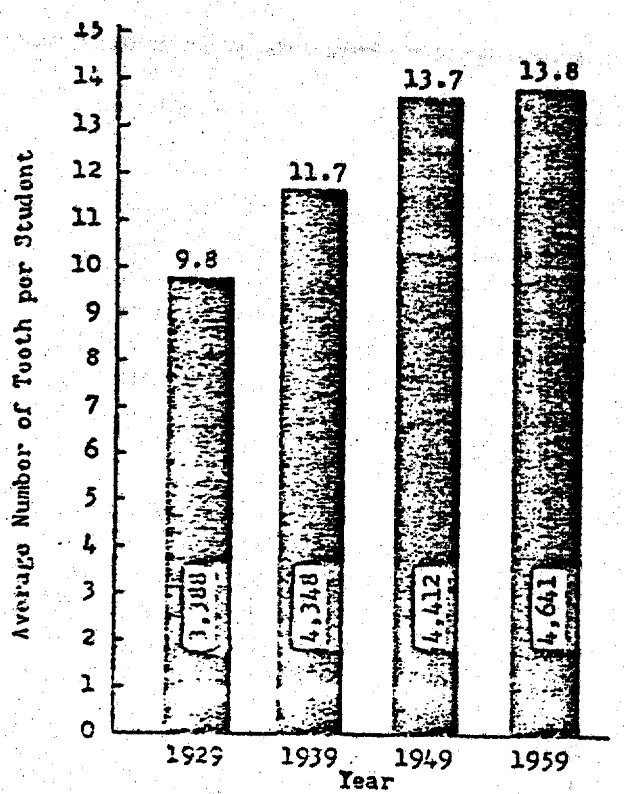

Fig 1 ■ Average number of teeth affected by caries in students entering University of Minnesota, 1929-1959 (Reproduced with author's permission.)

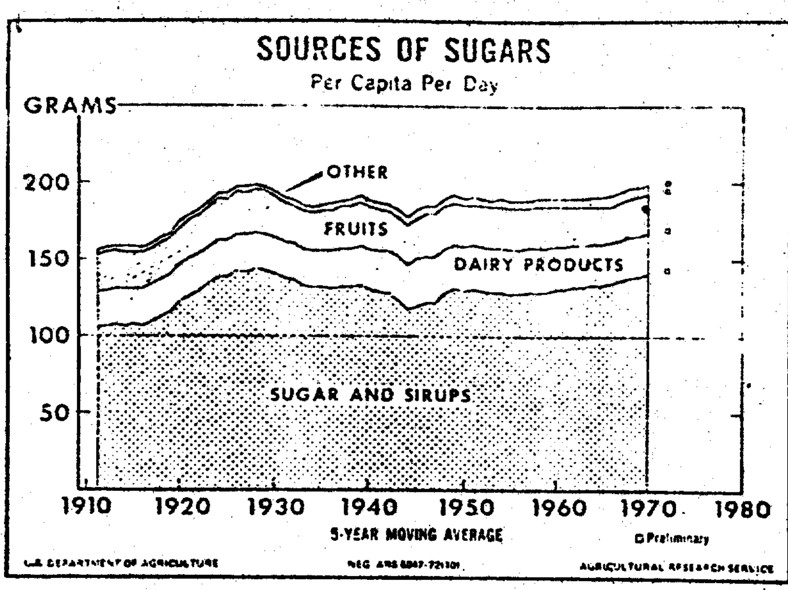

Fig 2 ▪ Per capita sugar consumption, 1910-1970. (Chart courtesy of investigators[9] and US Department of Agriculture.)

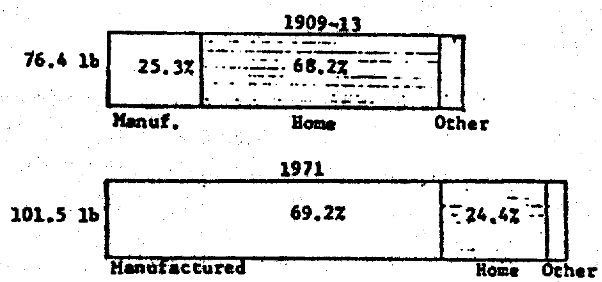

Fig 3 ▪ Per capita use of sugar (pound per capita) in manufactured products and by homemakers in 1910-1913 and 1971. (From US Department of Agriculture.[9])

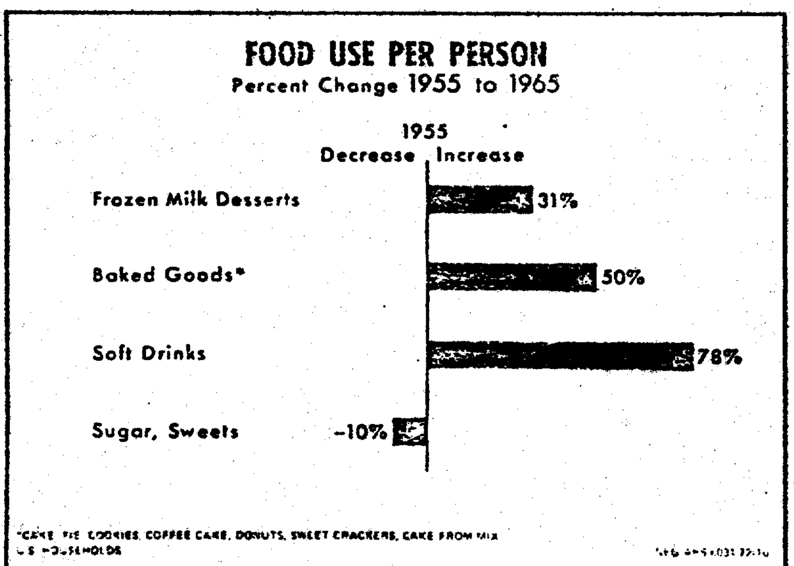

Fig 4 ▪ Changes in consumption of certain types of foods, 1955-1965. (Chart courtesy of investigators[9] and US Department of Agriculture.)

in almost all types of foods, but the amount that goes into carbonated beverages and snack foods has increased most rapidly.[9] Some relative increases in per capita consumption of sugar in different foods are shown in Figure 4 and Table 1. An increased use of snack foods can contribute to increased caries in two ways. First, the manufacturing processes increase the cariogenicity of sugars and starches or other constituents. Second, the availability and variety of snack foods, along with other social influences, have given rise to habits of more frequent eating that contribute to caries.

The second fact in support of my explanation is that, for one reason or another, the modern American is eating more frequently between

Table 1 ■ Pounds per capita consumption of sugar-containing foods and total sugar, 1925-1971

	1925-1929	1935-1939	1947-1949	1957-1959	1971	Change 1929-1971
Confectionery	8.0	8.2	9.8	9.4	11.0	37%
Cereal & bakery	7.7	9.7	12.9	15.4	17.6	130%
Processed vegetables	4.6	4.4	9.0	9.8	10.4	126%
Dairy products	2.3	2.4	4.6	4.9	5.8	152%
Beverages	5.0	5.2	10.6	12.6	22.8	356%
Total processed foods	28.4	31.1	48.4	50.0	70.2	105%
Total sucrose	100.0	97.1	94.8	95.4	101.5	1.5%

Data from Page and Friend.[9]

Table 2 ■ Changes in per capita servings of sweet (dessert) foods in 4,000 homes, 1963-1968.*

Food	Total %	Dessert %	Snacks %
Soft drinks	+32.5	+114.3	+26.3
Cakes	− 9.8	− 32.8	+70.4
Cookies	−11.9	− 42.6	+39.9
Fruit	−10.9	− 38.5	+56.1
Snacks (chips and so forth)	+28.1	+ 1	+63.2
Candy (chocolate)	+29.2	− 37.2	+46.5
Other candy	+41.0	− 8.3	+48.0

*An Economic and Marketing Report on Frozen Desserts, US Department of Commerce, October 1969.

meals and less regularly at set meal times. This may be of particular importance in respect to caries as indicated in Table 2, which shows that sugar-containing foods are used less frequently with meals[10] when they would be least destructive, and more frequently as snacks when they would be most damaging. Therefore, it seems likely that, as of today, the form and frequency with which sugar is used is more important than the amount eaten. Further, since the consumption of baked goods (Table 1) has increased in recent decades twice as rapidly as the consumption of more sugary confections, it seems logical to give more weight to such flour-sugar products as contributors to any increase in the strength of the caries attack that may have occurred.

Geography also has a relationship to caries prevalence. All known studies have indicated greater caries activity in the northeastern states of America than in the southern ones.[11] This is true even when factors such as fluorine content of the water and sociological factors are matched.[12] There is no parallel higher sucrose consumption in the northern states. Actually, in the low-caries southern region, the average weekly household (3.1 persons) consumption of sugar is higher (2.8 lb) than in the high-caries northeastern region (1.9 lb).[13] In contrast, US Department of Agriculture surveys[9,13] bring out that the consumption of baked goods containing flour and sugar is higher in the northern states (Fig 5) and that the spending for between-meal snacks is also higher (Table 3). However, car-

Fig 5 ■ Comparison of per capita consumption of baked goods in northern and southern states. (Chart courtesy of investigators[9] and US Department of Agriculture.)

Table 3 • Sugar and snack consumption, in pounds per household (3.1 persons) a week, in northeast (high caries) and southern (lower caries) states.

Region	Total sugar	Jams & jellies	Syrup	Drinks	Cola	Candy
Sugar						
Northeast	1.98	0.35	0.25	5.35	2.26	0.60
South	2.85	0.52	0.46	5.15	3.98	0.40
	Bread	**Baked goods**	**Crackers**	**Cakes**	**Cookies**	**Cost**
Snacks						
Northeast	4.85	3.62	0.59	0.64	0.93	$1.78
South	3.81	3.13	0.46	0.39	0.82	$1.30

Data from Research Service, US Department of Agriculture.[13]

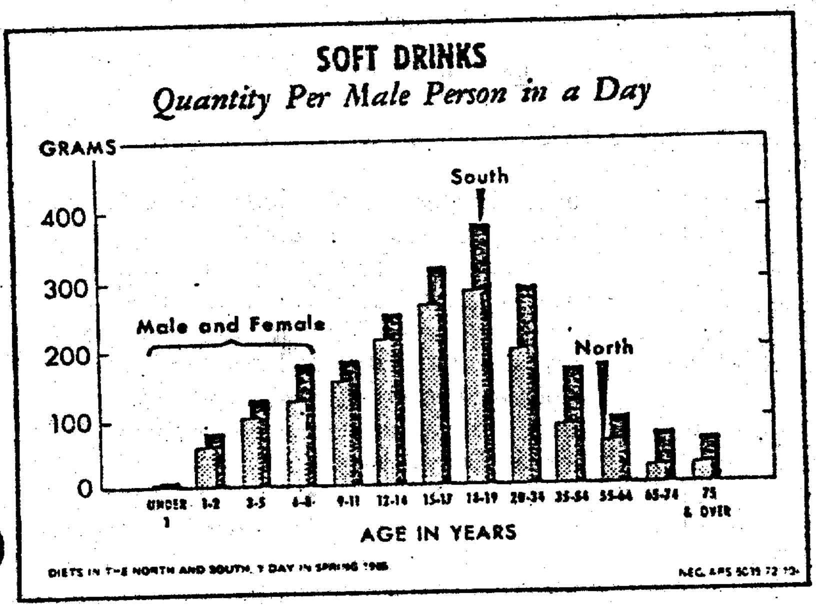

Fig 6 • Comparison of per capita consumption of soft drinks in northern and southern states. (Chart courtesy of investigators[9] and US Department of Agriculture.)

bonated beverage use is higher in the South[9] (Fig 6). All of this information supports the likelihood that differences in food composition, other than its sucrose content, are involved in caries causation and that such factors as food texture and the frequency of eating are important.

Information on caries prevalence and food use also is available for groups by sex. Girls have more permanent tooth caries than boys[14] of the same age although they eat less sugar and baked goods.[9] Earlier eruption of permanent teeth probably accounts for much of the higher caries rate in girls but more frequent snacking or other factors also may play a part.

Observations on selected human groups of limited size can be used to supplement broad epidemiologic findings. In an investigation of food relationships to caries, information on food use may be sought by study of persons whose caries state is known; conversely, caries assessments can be made after dietary modifications have been instituted. Studies of both sorts have been made.

A wide assortment of studies,[15-33] including one by Hyde, Bibby, and Brudevold, are in the former category. They vary in many aspects. One of these is the extent of the differences in caries between participants in high and low caries groups for which actual figures are not always given. Another is in the methods of information collection on the participants' diets; these methods ranged from parent interviews to the compilation of seven-day diet records. Since a full consideration of all of the variables is impossible, the essential information has been brought together (Table 4). The last column in the table, which lists the principal conclusions of the investigators, shows that the frequency of eating and high candy consumption are mentioned most often as causes of caries, followed closely by the use of flour-containing foods. Little evidence of nutritional effects is found. Only one study failed to associate high caries with one or another of these factors.

Studies of the other sort, in which the caries status was determined in persons on controlled

Table 4 ▪ Summary of reports on diets of high-caries and low-caries persons.

Authors	Date	Location	Caries groups High	Caries groups Low	Age	Authors' reasons for high caries
[illegible][15]	1934	Michigan	90	80	School	More desserts
[illegible][16]	1935	Various	19 racial groups		Various	More frequent eating, not nutrition
[illegible][17]	1936	Norway	40	11	School	More sweets and soft bread
Read & Knowles[18]	1938	England	12	12	3-14	Between meal eating, more sweets, and fermentable carbohydrates
Staz[19]	1943	South Africa	41	20	5-12	More frequent sweets
Hyde and others (unpublished data)	1945	New England	275 in 5 groups		Military	More meals and snacks
Bazant and others[20]	1954	Czechoslovakia	...	...	School	Sugar and white flour
Potgieter and others[5]	1956	Connecticut	864 in 3 groups		11-14	Poor diet, somewhat more between-meal eating, candy, and soft drinks
[illegible]dakara and others[21]	1956	Thailand	2,300 in 2 groups		4-18	More sugar and wheat flour
[illegible]enberger[22]	1958	Switzerland	249 in 2 groups		14-15	Higher sugar and white bread
[illegible]ecz[23]	1959	Hungary	3,859 in groups		3-6	Early weaning, high sweets, between-meal eating
Zita and others[24]	1959	Indiana	200 in 4 groups		5-13	More between-meal sugar
Weiss & Trithart[25]	1960	Tennessee	1,373 in 5 groups		5	More candies, cookies, and sodas
Bradford & Crabb[26]	1961	England	72	157	4-11	More carbohydrates
F[illegible] and others[27]	1962	Japan	808 in 4 groups		School	More cakes and candy after supper
MacGregor[28]	1963	Ghana	383 in 3 groups		6-12	More sweets
[illegible][29]	1969	Japan	155 subgrouped		2-3	More frequent eating
Palmer[30]	1971	England	366	355	7-10	Cariogenic food and liquid at bedtime
Samuelson and others[31]	1971	Sweden	1,401 subgrouped		4, 8, and 13	More sweets, buns, and cakes
Martinson[32]	1972	Sweden	156	167	14	Frequency of eating toffee, gum, and sweetmeats
Bagramian & Russell[33]	1973	USA	1,486 in 4 groups		High school	Not sucrose or frequency of eating

diets, do not give clear-cut results. Becks and co-workers[34] and Jay[35] found that elimination of sugar from the diet reduced caries activity in a majority of patients, but that often denial of starch-containing foods also was needed to stabilize the caries situation, as indicated by lactobacillus counts.

As part of a comprehensive study of the diets of children in three institutions in Pennsylvania, Mack and Urback[36] found the highest rate of caries (DMF/n, 0.341) in the children using 1 ounce of sugar a week, a lower rate (DMF/n, 0.242) in children using 2 ounces, and the lowest rate of caries (DMF/n, 0.17) in children using 3 ounces weekly. The diet of the institution with the lowest caries rate was nutritionally superior to diets at the other institutions. In Hopewood House, a residential institution in Australia, at which children used no refined sugar or flour, the number of decayed teeth was only about 6% of that in nearby Sydney children.[37]

In several studies, known amounts of sucrose have been added to the diets of institutionalized children. Mack and Urbach[36] had boys who lived in an institution with a good diet add a 2-ounce candy bar or its equivalent to their sugar intake each day for two years. No increase in caries was found, and the mean monthly increment of caries (DMF/n, 0.0032) was less than that usual for Pennsylvania boys. Koehne and Bunting[38] reported that the consumption of 3 lb of candy a week for five months produced active caries in 22 of 51 orphanage children who previously had low caries rates. In another study, Koehne and co-workers[39] added 100 g of sucrose in candy a day to the diet of 13 girls for periods of 5 to 18 months: they found that active caries developed in nine girls and that no caries developed in four. King[40] found that feeding two groups of young children 12.8 g of candy or 8.6 g of chocolate biscuit before bed each day for two years did not cause any caries. In another study, King and co-workers[41] added 35 to 71 lb of sugar a year to the diet of children in different institutions for periods of one or two years. The most definite caries increment was noted in molar fissures of 10- to 14-year-old children, but in some other situations there was less caries with the sugar supplements. The authors concluded that "relatively great differences in the total sugar content of the diets of children in institutions had no significant effect on the initiation or spread of dental caries in periods of one to two years," but "that it does not prove that the sugar content of the diet however distributed and however eaten never affects the teeth."

The best known and most comprehensive study of the effect of sucrose on caries was made in a mental institution in Sweden.[42] Sucrose, in amounts of from 10 to 120 kg a year, was fed to groups of from 39 to 62 adults, with meals, or between meals, or both, in liquid form or in bread or candies. Definite increases in caries resulted from the sugar additions, but these increases were not proportionate to the amount of sugar used. The frequency of eating and the vehicle in which the sugar was contained were of equal importance. For instance, the same 40 g of sugar eaten each day in caramels divided into four portions gave rise to almost twice as much caries as when it was divided into two parts. It was found that 30 g of sugar eaten in milk chocolate caused a mean of 1.35 lesions, whereas 40 g contained in caramels caused 3.55 lesions. Also 80 g of sugar in bread gave rise to a mean of 1.[illegible] lesions, whereas 330 g used with the same frequency in beverages caused only 0.43 lesions a year.

A recapitulation of information presented in the foregoing paragraphs serves to indicate its relationship to the question of updating dietary instruction for caries control. Although the strength of the caries attack seems to have increased during the past half century, there has been no corresponding increase in per capita sucrose consumption. This suggests that some other changes may relate more closely to the caries picture. Since the greatest change in eating habits in recent decades has been the increased use of manufactured, ready-to-eat snack foods, particularly baked goods, it seems logical to suspect them of being important in caries causation. This is particularly true since the increased variety and availability of such snack foods as well as other factors have led to an increase in the frequency of eating, which, of itself, is conducive to caries.

Support for the belief that snack food consumption is as important as the amount of sucrose used was found in a comparison of snack food and sugar use in the northeastern and southern states. Persons in the northeastern region, who have a higher caries incidence, eat more snacks but use less sugar than persons in the southern states, who have less caries.

Further reasons that suggest factors such as the frequency of eating or the use of the starchy or flour-sugar mixtures found in snacks may be as important as the total sugar intake are found in diet studies with small groups of humans. Although sugar obviously contributed to caries activity, a consistent relationship between the amount eaten and the corresponding caries response was not shown. Indeed, the best of these studies[42] indicated that the frequency of eating and the physical form in which sucrose was used were of equal or greater importance than the total amount eaten.

The only conclusion that can be drawn from the observations on humans is that factors other than sucrose intake seem to be important in caries causation. They also indicate that the frequency of eating is one of these factors and that starchy snack foods may be particularly damaging.

Animal caries

Since it was first demonstrated that coarse rice and corn would produce caries in rats, a tremendous variety of experimental animal diets has been shown to produce one type or another of carious lesions in rats, hamsters, and cotton rats. The gradual improvements in study methods, aimed at producing more consistent results in caries studies, have led step-by-step to questioning of the value of previous findings. This trend has climaxed in the realization that findings on rodent caries are of doubtful value unless the animals are in good health and both the amount of food eaten and the frequency of eating are controlled. This fact, plus recent demonstrations by Navia and co-workers[43] that the addition of as little as 5% of sugar or sugar alcohols to a starch-based diet will produce active caries, has convinced critical animal experimenters that, at this stage of their knowledge, most findings on rodent caries should not be regarded as having significance for man. Because of this, I find no point to presenting more than a brief summary of some findings of animal studies that might be paralleled in the human mouth.

Although many contradictory results are found in the mass of work that has been done on animals, the weight of the findings shows that sugar is more conducive to caries than starch[44-46]; that except under gnotobiotic conditions, it is uncertain whether sucrose is more conducive to caries than glucose[43,47-49]; that equal amounts of sugar in liquid or gel form produce only a fraction of the caries they produce in dry diets[47-50]; and that additions of fat can reduce caries production.[51,52] More recent work has demonstrated that the same total amount of food fed in many small portions will produce much more caries than that fed in fewer larger portions[53] and that gelatinized starches are much more cariogenic than raw starch.[54] In a caries workshop in September 1973, K. Madsen reported producing more caries in cotton rats with starch than with sucrose. Also, the amount of caries produced by human foods such as cookies or breakfast cereals has no direct relationship to their sugar content.[55-60]

In the only study in which a large number of foods of the sort eaten by man have been tested, Stephan[56] added to a rat diet a 66% portion of 53 candy and snack items. He found "that foods which were most cariogenic had a relatively high fermentable sugar content but this relationship was not necessarily quantitative." Natural sugars and fruits were found to be as cariogenic as refined sugars, and it was "important to recognize that starch alone may have cariogenic properties."

The use of monkeys,[61,62] rather than rodents, for caries studies offers promise of findings that could be interpreted with greater assurance to man. However beyond indicating that diets containing sucrose are cariogenic, the work with monkeys has cast no new light on the cariogenicity of human foods thus far.

In spite of the refinements that have been made in caries tests in animals, none of the findings can be taken safely as reliable evidence of the relative cariogenicity of foods for man. I can go no further than to concur with Stephan[56] that advice to avoid eating candy "may not be sufficient to control rampant caries because many other foods contain readily fermentable carbohydrates," and add that factors other than the amount of sucrose eaten influence animal caries and probably caries in man.

In vitro tests

In spite of the contributions that the in vitro experiments of early investigators made in establishing the importance of carbohydrates in caries causation, modern researchers have tended to discount their value in favor of the findings in rodent experiments. Now that we realize that the findings from such animal work have little relevance to the cariogenicity of foods in man, a case can be made for redirecting attention to in vitro studies. The usual methods for indicating the roles of different foods are measurements of their acid production, of enamel demineralization on incubation with oral bacteria, of food retention in the mouth after eating, and of acid formed in plaques on the tooth surface.

In other reviews,[63,64] I concluded that no single test method, of itself, gives a final answer as to the cariogenicity of foods but that combined information from several of them could be meaningful. Therefore, only the pertinent, previously cited information and some more recent findings are mentioned here.

▪ *Acid production:* Miller[65] differentiated between the caries-producing capacity of foods on the basis of the amount of acid they formed when incubated in saliva. He decided that bread and potatoes were more cariogenic than sugar because they formed more acid. Using a somewhat different procedure, Pickerill[66] reached the same conclusion. Beck and Bibby[67] pointed out defects in the experimental procedures of these early investigators, and by use of a procedure that neutralized accumulating acid before it inhibited glycolytic enzymes, we found that a number of foods produced more acid per unit of weight than did sucrose.

Bacterial growth and acid production also are influenced by sugar concentrations, a principle that is used in the preservation of many foods. My work with S. Mundorff (unpublished results) shows that this can apply to candies that yield up to a 20% solution of sugar in the mouth; at this level acid production is partly inhibited. Similarly, the acidity of foods prevents fermentation, and thus no new acid is formed on incubation in saliva of most sour-fruit flavored candies and beverages. It should be emphasized, however, that the inherent acid in such items will of itself produce enamel destruction in the mouth. Further, there is evidence[68] that flavor and other components of foods can modify, both quantitatively and qualitatively, the acids produced by fermenting foods.

▪ *Enamel demineralization:* Although it is generally assumed that the amount of enamel that would be destroyed by a fermenting food would be proportionate to the acid produced, this is not true.[69,70] Several factors account for this. First, as indicated by measurements of pH and titratable acidity, fermenting foods produce different spectra of acids that individually vary in their ability to dissolve enamel.[71] In addition, because of the content of minerals or proteins, many foods buffer acids formed during fermentation and thus reduce the effect of the acid on enamel. Further, some foods contain enamel-protective factors such as phytate identified by Jenkins[72] or the food proteins suggested by Weiss and Bibby[73] which are absorbed on the enamel, thereby making it more resistant to destruction by acid. It follows and has been shown experimentally[69,70,72] that there is no parallel between the sugar content of foods and the amount of enamel they will destroy in laboratory tests.

▪ *Food retention:* The amount of food retained in the mouth after eating has not been widely used as an index of cariogenicity. Although different methods of study have been used, it is agreed[74-76] that starchy foods persist about the teeth for longer periods and thereby will increase sugar retention. The unpublished findings of Bibby and Mundorff and the results of Rowle and co-workers[77] show that foods with high sugar content were removed more rapidly than those with less of such a freely soluble component. Thus, with sugary foods, retention was high immediately after eating but did not continue at that level unless the other food ingredients were of a retentive sort. Published[75] and our unpublished data show that fat in food tends to reduce its retention in the mouth. Liquid foods are removed more rapidly than dry ones, and carbonation seems to speed removal. Effects on salivary stimulation by acid or sugar content probably play a part in food clearance, but this is not clearly established.

▪ *Plaque pH:* Measurement of changes in plaque pH give information on the extent and duration of acid production from foods on the tooth surface and thereby should provide a direct indication of their cariogenicity. It is unfortunate that interpretation of plaque pH is made difficult by a lack of agreement in the findings obtained by different techniques of plaque pH measurement. The pH curves given by the telemetric method[78] differ in depth and duration from those given by the more generally used intraoral or extraoral procedures.[79-83] The latter show that glucose and sucrose have the same effect on plaque acidity. Smaller pH depressions are given by maltose and lactose and minimal changes by raw starch.[79] However, if starch is cooked, it gives much greater pH depressions,[79,80] and if it is used in association with sugar (in bread and cookies, for example), starch lowers the plaque pH to the same extent as sucrose[81] and maintains the depressed pH for a longer period.[82] Few studies[80,82-84] have tested more than one or two types of sugars or snack foods. The largest of these[84] showed that the patterns of pH change produced in plaques by food were influenced by the pH and buffering capacity of the foods as well as by their carbohydrate content and retention on the teeth. A clear relationship between plaque pH and the sugar content of foods was not apparent.

▪ *In vitro caries:* Although carieslike destruction of enamel has been produced in apparatus designed to duplicate oral conditions, such "artificial mouths" have provided little information on the cariogenicity of carbohydrates. One study[85] showed that glucose and sucrose demineralize enamel at the same rate, that lactose softens enamel less rapidly, and that soluble starch is essentially without effect. In a different type of apparatus, bread also produced carieslike enamel destruction.[86] In what I believe is a superior type of apparatus that has been developed,[87] we have produced enamel "caries" by more than 30 types of candies and snack foods. A direct relationship has not been found between "caries" production and the carbohydrate content of the foods tested; other variables obviously play a part.

▪ *Experimental in vivo caries:* Ethical and practical considerations have limited the number of attempts to use specific carbohydrates or foods to produce caries in the human mouth. Von der Fehr and co-workers[88] showed that fine white lesions developed on the teeth of patients who rinsed their mouths nine times a day for 23 days with a 50% sucrose solution and omitted oral hygiene. Using enamel blocks that were covered by gauze to hold "plaque" on them, Keller and associates[89] found as much enamel demineralization produced in the mouth by glucose as by sucrose. A description of a dental appliance on the teeth to retain acid or unspecified foods in contact with teeth also has been offered, but only acid seems to have been used to produce enamel destruction.[90] Bunting and coworkers[91] found that a gold cup, attached to the tooth surface and filled with bread, produced enamel caries in ten days.

The findings in the several types of tests reviewed in the preceding sections do not seem to point to any single type of food as being the most conducive to caries. In all of the test procedures, sucrose-containing items gave results that supported their ability to produce caries, but none of the findings pointed to a direct relationship between the amount of sugar in a food and its level of cariogenicity as indicated by the test method used. The acid production, demineralization, and in vivo caries tests that were done with nonsucrose materials indicated caries could be produced in the absence of sucrose. The results given by flour-containing foods in the demineralization, clearance, and plaque pH in vitro and in vivo caries experiments suggest that mixtures of flour and sugar may be particularly destructive to the teeth. In short, many variables in food composition—besides the carbohydrate or sugar content—can influence the results produced in in vitro tests and, by implication, the cariogenicity of foods. Among those that were pointed out are the existence of inhibitory levels of sugar or acid in foods, the presence of acid buffers or enamel protective agents, flavor effects, and food texture.

General discussion

The evidence on caries prevalence and on food use in man and evidence from animal and laboratory studies all indicate that variables other than the sucrose content of foods can be of importance in caries causation. The fact that more sucrose is eaten than any other carbohydrate does not displace sucrose as the major inciting agent of caries. As shown in the Vipeholm study,[42] the manner in which sucrose is used is of more importance than the total amount eaten. Many dentists and research workers have been guilty of oversimplification in assuming that a one-to-one relationship exists between sugar consumption and dental caries. The one certain conclusion at the present state of our knowledge is that several variables in food components, as well as those in oral biology, make it unlikely that any single type of food or food component can be named as the exclusive determinant of caries activity.

The conclusion that neither sucrose or any other specific type of carbohydrate can be named as a sole cause of caries is of theoretical rather than practical interest. Carbohydrates are not used in the pure state but as mixtures with water, flavoring agents, proteins, fats, or other carbohydrates. Thus, even if results had shown that sucrose alone gives rise to caries, they would have had limited usefulness in combating dental caries unless we also knew whether this sugar was equally cariogenic in all concentrations, regardless of its patterns of use or what it might be mixed with in foods and beverages. In other words, what is needed to give practical advice to patients on caries prevention is not information on the relative cariogenicity of pure carbohydrates, but data on the cariogenicity of the actual food items that they eat. Furthermore, since we know that between-meal or snack eating is particularly conducive to caries and that the use of snack foods is increasing, it follows that the most needed information for diet counseling is on the relative cariogenicity of those carbohydrate-containing foodstuffs, candies, snack foods, and beverages that are most commonly used between meals.

In this connection, attention should be directed to foods made of mixtures of flour and sugar. Laboratory tests and some animal studies indicate that these may be particularly destructive to the teeth. Also, this type of baked-goods snack food is showing the greatest increase in use in this country. Further, since these items are designed for between-meal use, they can be counted on to exert their maximum destructive effect on the teeth.

The value of knowing which snack foods are highly destructive to the teeth and which have less harmful effects becomes more important if one believes, as I do, that between-meal eating—the principal activating cause of caries—is a practice that, because of social, industrial, and medical influences, is going to become progressively harder for dentists to combat. Therefore, if we as dentists have to live with an increasing use of snack foods, then it is our responsibility to make the habit as harmless to the teeth as possible. The only way to do this will be to tell patients which foods are highly cariogenic and suggest foods of low cariogenicity that they should use in their diet. Also, if such low cariogenic substitutes cannot be found on the market, then research workers and manufacturers should be encouraged to develop them and make them available to the public. This is not an unrealistic possibility.

Several approaches to finding foods of low cariogenicity are possible. One of these is to find additives such as the phosphates, which make highly cariogenic foods less destructive to the teeth. A second is to sweeten foods with saccharin, cyclamate, or other noncaloric sweeteners so that the sugar content of foods can be reduced or eliminated. The third is to examine foods that are already in use to determine whether sufficiently great differences in their cariogenicities justify the recommendation of some available foods in place of those with a higher cariogenicity. The advantage of the last approach is that it could eliminate problems of developing new formulations and manufacturing and distribution methods that present far greater problems than are realized by those who are not food manufacturers.

A low cariogenic food has to be competitive in taste appeal and cost to be useful. Therefore, pending developments of a line of food products containing protective additives or sugar substitutes, it seemed worthwhile to evaluate by a variety of methods the cariogenicity of a wide spectrum of available snack foods, confections, and beverages to determine which of them might be mildly destructive; a second objective was to explain the differences in cariogenicity that have been reported to exist between similar types of food. Our investigations, which will be reported elsewhere, indicate that foods do give evidence of wide differences in cariogenicity, aside from their sugar content. Along with the material presented here, they also suggest that attention should be given to factors other than the sucrose content of foods in the diet counseling of patients.

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1. Hardwick, J.L. The incidence and distribution of caries throughout the ages in relation to the Englishman's diet. Br Dent J 108:9 Jan 1960.

2. Sedwick, H.J. Observations on pre-Columbian Indian skulls unearthed in New York State. JADA 23:764 May 1936.

3. Sognnaes, R.F. Further analysis of war time caries observations. Br Dent J 87:291 Dec 1949.

4. Toverud, G. Survey of the literature of dental caries. Washington, National Research Council, 1952.

5. Takeuchi, M. Epidemiological study on relation between dental caries incidence and sugar consumption. Bull Tokyo Dent Coll 1:58 Oct 1960.

6. Bartholdi, W.L.; Petersen, E.E.; and Crawford, W.H. University of Minnesota freshmen student survey. JADA 63:868 Dec 1961.

7. Glass, R.; Chiang, T.P.; and Fleisch, S. Secular trends in caries prevalence. Abstracted, IADR Program & Abstracts no. 677 March 1972.

8. Potgieter, M., and others. The food habits and dental status of some Connecticut children. J Dent Res 35:638 Aug 1956.

9. Page, L., and Friend, B. Level of use of sugars in the United States. Read before the International Conference on Sugars in Nutrition, Vanderbilt University, 1972.

10. US Department of Commerce. An economic and marketing report on frozen desserts. Prepared for the International Association of Ice Cream Manufacturers, Oct 1969.

11. Dunning, J.M. Incidence and distribution of dental caries in the US. Dent Clin North Am July 1962, p 291.

12. Ludwig, T.G., and Bibby, B.G. Geographic variations in the prevalence of dental caries in the United States of America. Caries Res 3:32, no. 1, 1969.

13. Food consumption of households in the United States. US Department of Agriculture, reports no. 2 & 4, 1956.

14. Finn, S.B. Survey of the literature of dental caries. Washington, National Research Council, 1952.

15. Pepin, C., and Miller, H.I. A comparison of the incidence of caries with the diet of two groups of Michigan school children. J Mich State Dent Soc 16:569, 1934.

16. Bibby, B.G. Neglected factors in the study of dental caries. JADA 22:222 Feb 1935.

17. Collett, A. Diet and dental caries. Lancet 1:36 Jan 4, 1936.

18. Read, T.G., and Knowles, E.M. A study of the diets and habits of school children in relation to freedom from or susceptibility to dental caries. Br Dent J 64:185, 1938.

19. Staz, J. Dental caries in Johannesburg Indian children. South African Dent J 17:152 May 1943.

20. Bazant, V.; Miska, J.; and Skopkova, M. Dental caries and nutrition in the region of Sedlany, Radonik and Prague in 1948-49. In Brislin, J.F., and Cox, G.J., eds. Survey of literature of dental caries. Pittsburgh, University of Pittsburgh Press, 1964, p 279.

21. Kridakara, O., and others. Dental survey of selected Thai children. Am J Clin Nutr 4:230 May-June 1956.

22. Eppenberger, K. Caries prevalence in the Toggenberg valley. Helv Odontol Acta 1:1 April 1958.

23. Oravecz, P. Role of diet and oral hygiene in prevalence of decay of kindergarten children in the county Baranya. Forgorvas Szemle 52:32 no. 1, 1959.

24. Zita, A.C.; McDonald, R.E.; and Andrews, A.L. Dietary habits and the dental caries experience in 200 children. J Dent Res 38:860 Sept-Oct 1959.

25. Weiss, R.L., and Trithart, A.H. Between meal eating habits and dental caries experience in pre-school children. Am J Public Health 50:1097 Aug 1960.

26. Bradford, E.W., and Crabb, H.S.M. Carbohydrate restriction and caries incidence. Br Dent J 111:273 Oct 1961.

27. Fukui, T.; Myosi, T.; and Endo, K. Investigation on the relation of between-meal eating and dental caries of sixth year molars in school children. Shikoku Acta Med 18:392 no. 5, 1962.

28. MacGregor, A.B. Increasing caries incidence and changing diet in Ghana. Int Dent J 13:516 no. 3, 1963.

29. Niwa, T. Between meal snacks and their relationship to dental caries in small children. Jap J Dent Health 19:1 no. 1, 1969.

30. Palmer, J.D. Dietary habits at bedtime in relation to dental caries in children. Br Dent J 130:288 April 1971.

31. Samuelson, G.; Grahnen, H.; and Arvidsson, E. An epidemiological study of child health and nutrition in a northern Swedish county. Am J Clin Nutr 24:1361 Nov 1971.

32. Martinson, T. Socio-economic investigation of school children with high and low caries frequency. Odontol Revy 23:93 no. 1, 1972.

33. Bagramian, R.A., and Russell, A.L. Epidemiologic study of dental caries experience and between meal eating patterns. J Dent Res 52:342 March-April 1973.

34. Becks, H.; Jensen, A.L.; and Millarr, C.E. Rampant dental caries: prevention and prognosis. A five year clinical survey. JADA 31:1189 Sept 1944.

35. Jay, P. The reduction of oral Lactobacillus acidophilus counts by the periodic restriction of carbohydrate. Am J Orthod Oral Surg 33:162 March 1947.

36. Mack, P.B., and Urbach C. A comparison of medical, dental and laboratory observations on three groups of institution children on dietaries which differed in caloric intake and in level of intake of certain nutrients. Mono Sec Res Child Devel 13:1, 1947.

37. Lilienthal, B., and others. The biology of the children of Hopewood House, Bowral, N.S.W. Med J Aust 2:878 Dec 1953.

38. Koehne, M., and Bunting, R.W. Studies in the control of dental caries. J Nutr 7:657 no. 6, 1934.

39. Koehne, M.; Bunting, R.W.; and Morrell, E. Control of dental caries in children. Am J Dis Child 48:6 no. 1, 1934.

40. King, J.D. Dental caries. Effect of carbohydrate supplements on susceptibility of infants. Lancet 1:646 June 1946.

41. King, J.D., and others. The effect of sugar supplements on dental caries in children. Med Res Council special report no. 288, 1955.

42. Gustafsson, B.E., and others. The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. Acta Odontol Scand 11:232 no. 3-4, 1952.

43. Navia, J.M.; Lopez, H.; and Fischer, J.S. Caries promoting properties of sucrose substitutes in foods: mannitol, xylitol and sorbitol. J Dent Res 53 (special issue):207 abstract no. 611 Feb 1974.

44. McClure, F.J. Dietary factors in experimental rat caries and advances in experimental caries research. Washington, American Association for the Advancement of Science, 1955.

45. Navia, J.M. Evaluation of nutritional and dietary factors that modify animal caries. J Dent Res 49:1213 (suppl) Nov-Dec 1970.

46. König, K.G., and Grenby, T.H. The effect of wheat grain fractions and sucrose mixtures on rat caries developing in two strains of rats maintained on different regimes and evaluated by two different methods. Arch Oral Biol 10:143 Jan 1965.

47. Constant, M.A.; Phillips, P.H.; and Elvehjem, C.A. Dental caries in the cotton rat. XII. Natural verus refined sugars. J Nutr 43:551 April 1951.

48. Green, R.M., and Hartles, R.L. The effects of diets containing different mono and disaccharides on the incidence of dental caries in the albino rat. Arch Oral Biol 14:235 March 1969.

49. Shaw, J.H. Influence of experimental diets on carious lesions in the occlusal sulci and smooth surfaces of the molars of susceptible rats. J Dent Res 52:291 March-April 1973.

50. Harris, M.R. and Stephan, R.M. Effect of mixing water in the diet on development of carious lesions in rats. J Dent Res 32:653 abstract no. 50 Oct 1953.

51. Shaw, J.H. Effects of dietary composition on tooth decay in the albino rat. J Nutr 41:13 May 1950.

52. Gustafsson, G., and others. Experiments with various fats in a cariogenic diet. Acta Odontol Scand 13:75 no. 2, 1955.

53. König, K.G. Caries activity induced by frequency-controlled feeding of diets containing sucrose or bread to Osborne-Mendel rats. Arch Oral Biol 14:991 Aug 1969.

54. Frostell, B., and Baer, P.N. Caries experience of rats fed various starches for study of experimental calculus formation. Acta Odontol Scand 29:401 no. 4, 1971.

55. Zepplin, M., and others. Dental caries in the cotton rat. The effect of feeding a natural diet comparable to a human diet. J Nutr 40:203 Feb 1950.

56. Stephan, R.M. Effect of different types of human foods on dental health in experimental animals. J Dent Res 45:1551 Sept-Oct 1966.

57. Ishii, T.; König, K.G.; and Mühlemann, H.R. The cariogenicity of different between-meal snacks in Osborne-Mendel rats. Helv Odontol Acta 12:41 Oct 1968.

58. Hartles, R.L. Carbohydrate consumption and dental caries. Am J Clin Nutr 20:152 Feb 1967.

59. Choung, U.B.; Bibby, B.G.; and Losee, F.L. Some effects of breakfast cereals on caries in rats. J Dent Res 52:504 May-June 1973

60. Stookey, G.K., and Muhler, J.C. Anticariogenic effect in the rat of cereals fortified with soluble phosphate. J Dent Res 45:856 May-June 1966.

61. Bowen, W.H. The induction of rampant dental caries in monkeys (Macaca iris). Caries Res 3:227, no. 3, 1969.

62. Bowen, W.H. The cariostatic effect of calcium glycerophosphate in monkeys. Caries Res. 6:43, no. 1, 1972.

63. Bibby, B.G. Local effects of nutrients. Symp Swed Nutr Found 3:30, 1964.

64. Bibby, B.G. Methods for comparing the cariogenicity of foods. J Dent Res 49:1334 (suppl) Nov-Dec 1970.

65. Miller, W.D. Microorganisms of the human mouth. Philadelphia, S.S. White Publishing Co., 1890.

66. Pickerill, H.P. The prevention of dental caries and oral sepsis. London, Bailliere, Tindall & Cox, 1912.

67. Beck, D.J., and Bibby, B.G. Acid production by the fermentation of starches by saliva. J Dent Res 40:486 May-June 1961.

68. Mundorff, S., and Bibby, B.G. Enamel dissolution by snack foods. J Dent Res 52 (special issue):266 abstract no. 829 Feb 1973.

69. Andlaw, R.J. The relationship between acid production and enamel decalcification in salivary fermentations of carbohydrate foodstuffs. J Dent Res 39:1200 Nov-Dec 1960.

70. Weiss, M.E., and Bibby, B.G. Enamel dissolution by streptococcal fermentation of breakfast cereals. J Dent Res 49:1481 Nov-Dec 1970.

71. Buonocore, M.G. Dissolution rates of enamel and dentin in acid buffers. J Dent Res 40:561 May-June 1961.

72. Jenkins, G.N. Enamel protective factors in food. J Dent Res 49:1318 (suppl) Nov-Dec 1970.

73. Weiss, M.E., and Bibby, B.G. Some protein effects on enamel solubility. Arch Oral Biol 11:59 Jan 1966.

74. Lundquist, C. Oral sugar clearance. Odontol Revy 3: (suppl 1) 1 1952.

75. Bibby, B.G.; Goldberg, H.J.V.; and Chen, E. Evaluation of caries-producing potentialities of various foodstuffs. JADA 42: 491 May 1951.

76. Swenander-Lanke, L. Oral carbohydrate clearance. Symp Swed Nutr Found 3:53, 1964.

77. Rowley, J.E.; Bibby, B.G.; and Mundorff, S. Oral retention of carbohydrate foods. J Dent Res 52 (special issue):269 abstract no. 842 Feb 1973.

78. Muhlemann, H.R., and de Boever, J. Radiotelemetry of the pH of interdental areas exposed to various carbohydrates. In McHugh, W.D., ed. Dental plaque. Dundee, Scotland, D.C. Thomson & Co., Ltd., 1970.

79. Neff, D. Acid production from different carbohydrate sources in human dental plaque in situ. Caries Res 1:78 no. 1, 1967.

80. Frostell, G. Effect of a cooked starch on the pH of dental plaque. Swed Dent J 65:161 no. 2, 1972.

81. Kleinberg, I., and Jenkins, G.N. Further studies in the effect of carbohydrate substrates on plaque pH in vivo. J Dent Res 38:704 July-Aug 1959.

82. Ludwig, T.G., and Bibby, B.G. Acid production from different carbohydrate foods in plaque and saliva. J Dent Res 36:56 Feb 1957.

83. Mörch, T. The acid potentiality of carbohydrates. Acta Odontol Scand 19:355 no. 3-4, 1961.

84. Edgar, W.M., and others. Acid production in plaques after eating snacks: modifying factors in foods. JADA, in press.

85. Pigman, W.; Brasher, J.; and Koulourides, T. Cariogenicity of common sugars as evaluated in the artificial mouth. Nature 195:190, July 1962.

86. Dietz, V.H. Production of plaques and caries. J Dent Res 22:423 Dec 1943.

87. Yaari, A., and Bibby, B.G. Apparatus using thawing human saliva for in vitro caries study. J Dent Res 52 (special issue):93 abstract no. 139 Feb 1973.

88. Von der Fehr, F.R.; Löe, H.; and Theilade, E. Experimental caries in man. Caries Res 4:131 no. 2, 1970.

89. Keller, S., and others. Supplementary sucrose vs glucose effect on human experimental caries. J Dent Res 52 (special issue):265 abstract no. 827 Feb 1973.

90. Nygaard-Ostby, B.; Hals, E.; and Mörch, T. A method for experimental in vivo decalcification of dental enamel. Acta Odontol Scand 15:347 no. 4, 1957.

91. Bunting, R.W.; Nickerson, G.; and Hard, D.G. Further studies of the relation of Bacillus acidophilus to dental caries. Dent Cosmos 68:931 Oct 1926.

Considerable differences were found in the extent and duration of pH fall in plaque after the use of 54 snack foods. These differences made it possible to rank the foods in order of acidogenicity (and likely cariogenicity). Parallel measurements of food retention, carbohydrate concentration in saliva, and saliva flow brought out some correlation but no consistent overall relationships among the several parameters. Plaque acidity is the result of extraneous food acids entering plaques as well as fermentation acids formed in them.

Acid production in plaques after eating snacks: modifying factors in foods

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The frequency of consumption of carbohydrate foods and their continuing presence in the mouth after eating are known to be important causes of caries (Gustafsson and co-workers[1] and Lundquist[2]), probably by reason of their effects on acid production in plaques where tooth destruction is initiated. Because little is known about these relationships, an investigation on the extent and duration of the depressions in plaque pH produced by different foods and beverages was undertaken. It is believed that the information obtained could serve as an index of the cariogenicity of foods and that it could be used in dietary counseling for patients unable or unwilling to eliminate between-meal eating.

Previous work in this field by Kleinberg[3] has given insight into the complexity of the diet-plaque-caries relationship, but has provided only limited information relating the properties of different foods to the changes they provoke in plaque pH. Accordingly, factors such as the rate of clearance of the food from the mouth, its pH and carbohydrate concentration in saliva, and its ability to stimulate a protective salivary response were studied in parallel with plaque pH measurements.

Materials and methods

■ *Plaque pH measurements:* Studies were made on 19 young adults on whose teeth significant plaque formation occurred. They continued to use their ordinary diets and did not clean their teeth for 24 hours, or take food or drink for 2.5 hours before each experimental session. As far as possible these were at the same time each day.

Plaque pH was determined by a method similar to that used by Fosdick and co-workers,[4] Ludwig and Bibby,[5] and Frostell.[6] Plaque samples were taken from all accessible surfaces of premolars and molars with a stainless steel microspatula. These were mixed with a drop of 0.45% sodium chloride solution in a one-drop glass electrode,* having a calomel half-cell with fiber-liquid junction.† With an expanded scale, a pH reading with 0.01 units accuracy was obtained 90 seconds after sampling. The electrode system was standardized with buffers at pH 4.00, 5.00, and 7.00 each day and checked between each experimental session. Plaque sampling and pH determination were done before, immediately after, and at 5, 10, 15, 20, and 30 minutes after consumption of a test food.

■ *Saliva pH and flow rate:* Saliva was collected over a one-minute period before and between 1 and 2, 6 and 7, 11 and 12, and 16 and 17 minutes after the food was taken. The study participants were instructed to sit quietly and either to allow the saliva to drool from the mouth or to spit out the saliva collecting in the mouth during the one-minute period. They used the same collection technique at each experimental session.

The saliva was collected in preweighed polyethylene containers and immediately a sample weighing 5 to 10 mg withdrawn for pH measurement on the one-drop electrode. The remaining sample of saliva was weighed in its container to an accuracy of 0.01 g and the weight of saliva secreted in one minute calculated.

■ *Food retention:* The method used was based on preliminary trials. Each of three participants consumed a standard portion (or a normal mouthful) of the test snack. At five minutes after normal deglutition and completion of the associated physiologic movements of tongue and cheek, the participant rinsed his mouth for 15 seconds with 15 ml of water, brushed the teeth and gingiva with a wet brush according to his established practice, and then rinsed once more as before. All rinsings and the washings from the toothbrush were collected in a beaker and stored frozen until analyzed. The test was repeated, with new samples of food, with intervals of 15 and 30 minutes between food use and rinsing. The carbohydrate content of each rinsing was determined by the anthrone method of Beck,[7] which also was used for measurement of the amount of soluble and insoluble carbohydrate in each food. From these data, the amount (in glucose equivalents) of soluble, insoluble, and total carbohydrate and total food could be derived. The findings were recorded as the mean value for the three persons.

■ *Snack foods tested:* A total of 54 foods, excluding glucose rinses used for standardization, were tested. Information on these foods is given in Table 1. For the food retention studies where certain candies were available in more than one flavor, only one flavor was tested. With a few exceptions, the foods were purchased from a single retail outlet.

■ *Snack food use:* Different groups of foods were selected for the plaque pH experiments: hard (sucking) candies, soft candies and gums, fruits and beverages, and baked goods. The foods in each group were tested by five participants who composed groups that were matched for sex (two women, three men), their resting plaque pH, and pH change after two-minute rinses with a 10% glucose solution.

Although every attempt was made to maintain a normal pattern of food use, the variety in types of test foods required a degree of standardization to permit comparisons to be made. Hard candies were sucked for five minutes, at which time the partially dissolved candy was removed from the mouth and weighed with its wrapper. The difference between this and the initial weight gave a measure of the weight of candy consumed. The portions of soft candies and chocolate, one candy or approximately 10 g of candy bars, were consumed within five minutes. For them the time taken for eating was noted. Gums were chewed for five minutes, although the flavor, and presumably most of the soluble carbohydrate was reported to be lost after two to three minutes. Cookies and crackers were given as one portion and as two normal mouthfuls. This was also done for fruits. Raisins and nuts were offered as 10-g portions and beverages as 100-ml portions. Drinking time was noted.

With all foods except beverages, the participants were instructed to chew or suck the food normally, but to inhibit swallowing and to spit the food-saliva mixture into a preweighed beaker. After mixing had been completed, the pH and weight of the spat material were determined. The known weight of food eaten was subtracted from the weight of the mixture and, hence, the mean carbohydrate concentration of the environment of the plaque during food use could be estimated. It was impossible to recover all of the beverage as an expectorate, but because of its much greater volume, it is not thought that the saliva would modify to any degree its pH or concentration in the mouth.

Findings

■ *Plaque pH results:* In most instances the plaque pH values followed biphasic curves of the type described by Stephan[8]; differences between individuals and between foodstuffs appeared in the shape and extent of the pH depressions. Two parameters were selected to analyze these differences: the pH minimum reached after

Table 1 ■ Snack foods tested.

Name	Remarks and constituents of interest
Hard candies	
Suckers	Raspberry, cherry flavored lollipops, clear, hard candy. Principally sucrose.
Sourballs	Grape, orange flavored, clear hard candy. Sucrose, glucose, citric, and malic acids.
Rock candy	Crystallized sucrose; light or dark color.
Sweetees*	Clear hard candy. Sorbitol 79.7%, gum arabic 10%, citric acid.
A-plus*	Clear hard candy. Sorbitol 30%, lycasin (hydrogenated starch product) 68%, citric acid.
Clear mints	Clear hard candies. Principally sucrose.
Soft candies	
Ju-ju Toys*	Gum candy; fruit flavors. Sucrose, corn syrup, starch, citric acid.
Giant Jellies*	Sugar-coated gum candy, fruit flavors and licorice. Sucrose, corn syrup, starch.
Licorice Drops*	Semisoft gum candy. Corn syrup, sucrose, starch, shortening, gelatin.
Licorice Bites*	Soft gum candy. Flour, sucrose, corn syrup, licorice mass, burnt sugar.
Chocolate	Milk and semisweet flavors.
Sugar-coated gum	Sugar-coated chewing gum tablets. Chewing gum base, sugar, corn syrup, modified food starch.
Trident sugarless gum*	Sugarless chewing gum slabs. Chewing gum base, sorbitol, mannitol, saccharin.
Dentyne*	Chewing gum slabs. Chewing gum base, sucrose, corn syrup.
Caramel	Condensed skim milk, corn syrup, sucrose, vegetable oil, cream, whey solids.
Baked goods	
White bread	Plain white bread. Flour, shortening, yeast, malt, monoglycerides, calcium propionate. Unsalted butter. Grape flavored jelly.
Sweetened bread	Enriched wheat flour; honey.
Rye bread	Wheat and rye flours, shortening, malt, caramel.
Whole wheat bread	Whole wheat and white flours, corn syrup, shortening, milk powder, malt, honey, calcium propionate.
Doughnuts	Plain.
Bagels	High gluten flour, sucrose, whole egg solids, malt, calcium propionate.
Oil-sprayed crackers	Enriched four, malt.
Unsalted crackers	Enriched flour.
Saltine crackers	Enriched flour, malt.
Graham crackers	Enriched wheat flour, graham flour, sucrose, molasses.
Chocolate grahams	Enriched flour, graham flour, sucrose, cocoa powder, nonfat milk solids, honey, molasses.
Sponge cake	Plain sponge without glazing. Sucrose, egg yolks, whole eggs, flour, nonfat dry milk.
Twinkies*	Filled sponge cakes. Sucrose, flour, eggs, corn sugar, whey, nonfat dry milk, soy flour, corn syrup, starch.
Astrofood*	Filled high-protein sponge. Sucrose, flour, calcium caseinate, corn syrup, eggs, nonfat dry milk, raspberry puree, dicalcium phosphate, corn starch, agar, sorbitan monostearate.
Pound cake	Sucrose, whole eggs, enriched flour, whole milk.
Angel food cake	Egg whites, sucrose, flour, starch, cream of tartar.
Chocolate cake	Sucrose, enriched flour, whole eggs, chocolate, cocoa, corn syrup, whole milk, buttermilk.
Apple pie	Crust: flour, glucose, baking soda. Filling: apples, sucrose, corn starch.
Plain cookies	Enriched wheat flour, corn flour, corn syrup, eggs, sucrose.
Sandwich cookie	Cream-filled. Filling: soya protein, nonfat dry milk, rye flour, egg white, lecithin.
Beverages and other foods	
Coca-Cola*	10.5% carbohydrate.
7 up*	10.3% carbohydrate.
Orange juice	Whole orange juice, unsweetened.
Apple juice	Unsweetened.
Milk	Fortified whole milk.
Chocolate milk	Skimmed milk, whole milk, sucrose, chocolate.
Potato chips	Potatoes, vegetable oil, salt.
Peanuts	Shelled, roasted.
Cereal	Cap'n Crunch:* corn and oat flours, sucrose, brown sugar.
Apple	Red Delicious.
Banana	...
Raisins	Seedless.
Dates	...

*Trade name.

food use and the sum of all pH depressions below the fasting baseline level (ΣD). This latter value was found to be closely correlated with the triangulated area of the curve below a line drawn horizontally from the baseline and was used as an index that reflected the duration as well as the severity of the pH fall.

The data obtained for the glucose rinse procedure were subjected to analysis of variance, and no significant difference between means for the individuals between groups could be found either for pH minimums or ΣD values; this suggests that the individuals were well matched for the purpose of comparison of the plaque pH changes in response to foods in different groups.

So that the results could be displayed in a useful form, the mean values found for five individuals for each food were ranked in order of pH minimum and ΣD values. Those foods with high pH minimum or low ΣD values were given low

Table 2 ▪ Effect of snack foods on plaque pH minimum; foods ranked from least to most acidogenic.

Rank	Food	pH minimum*	ΣD†	ΣD rank†
1	Trident sugarless gum	6.62	−0.46	1
2	Peanuts	6.57	0.09	2
3	Milk	6.38	0.75	4
4	Licorice Giant Jellies	6.17	2.14	13
5	Licorice Bites	6.14	1.50	7
6	Dentyne gum	6.11	2.55	19
7	Dark chocolate	6.10	1.98	10
8	Apple	6.09	1.80	8
9	Potato chips	6.08	2.04	11
10	White bread	6.07	1.20	5
11	Milk chocolate	6.06*	2.35	15
12	Licorice Drops	6.06*	2.98	29
13	Chocolate milk	6.01	2.49	17
14	Sugar-coated gum	5.98	2.45	16
15	Caramel	5.95*	2.85*	22
16	Bread & butter	5.95*	2.10	12
17	Saltine crackers	5.89	2.69	20
18	Bagel	5.88	3.24	34
19	Banana	5.87	2.94*	26
20	Sweetees	5.86	0.66	3
21	Whole wheat bread	5.85	3.68	43
22	Dates	5.82	3.39	38
23	Bread & jam	5.81	3.70*	44
24	Graham crackers	5.80*	1.49	6
25	Orange Giant Jellies	5.80*	3.04	30
26	Oil-sprayed crackers	5.79*	2.26	14
27	Unsalted crackers	5.79*	2.87	24
28	Coca-Cola (as drink)	5.78	3.20	33
29	Twinkies	5.76	2.52	18
30	7 up	5.74	3.10	31
31	Doughnut	5.68*	2.96	28
32	Pound cake	5.68*	3.49	39
33	Cereal	5.65	3.35	36
34	Sandwich cookies	5.63*	2.85*	23
35	Apple pie	5.63*	4.17	49
36	Apple juice	5.62	2.94*	27
37	Astrofood	5.61	3.38	37
38	Sponge cake	5.60*	3.98	47
39	Chocolate cake	5.60*	3.60	41
40	Plain cookies	5.59	2.79	21
41	Rock candy (dark)	5.55	3.31	35
42	Rock candy (light)	5.52*	3.91	46
43	Mint candy	5.52*	4.12	48
44	Orange juice	5.52*	3.11	32
45	Coca-Cola (as rinse)	5.50	4.25	50
46	Raisins	5.48	2.93	25
47	Chocolate graham crackers	5.45*	3.86	42
48	Angel food cake	5.45*	4.48	54
49	Raspberry sucker	5.39*	3.70*	45
50	Ju-ju Toys	5.39*	4.45	53
51	A-plus (lycasin)	5.36	1.81	9
52	Orange sourballs	5.33	4.30	51
53	Grape sourballs	5.30	4.42	52
54	Cherry sucker	5.22	3.59	40

*Where necessary, mean pH values were calculated to three decimal places to break tied ranks.
† ΣD is the sum of all pH depressions below resting pH level. It indicates the duration as well as the severity of the pH fall. In ΣD rank column, lowest figures represent smallest pH change.

rank numbers, and those that give rise to a greater or more prolonged pH fall were given high rank numbers (Table 2). The rankings for the two parameters were closely correlated, as shown by Spearman's rank correlation coefficient ($r_s=0.74$; $P<0.001$).[9]

For a majority of foods, ranking for the two parameters did not differ by ten ranks or more. These foods were considered to provoke a normal pattern of fall in plaque pH (type A curve). Discrepancies in ranking of ten ranks or more seemed to identify foods that provoke an unusual pattern of pH fall; where the rank for pH minimum was lower than the ΣD value (type B curve) a prolonged or delayed pH fall might be expected, whereas where the pH minimum rank was higher than the ΣD rank (type C curve) the pH fall might be expected to be brief. Figure 1 shows

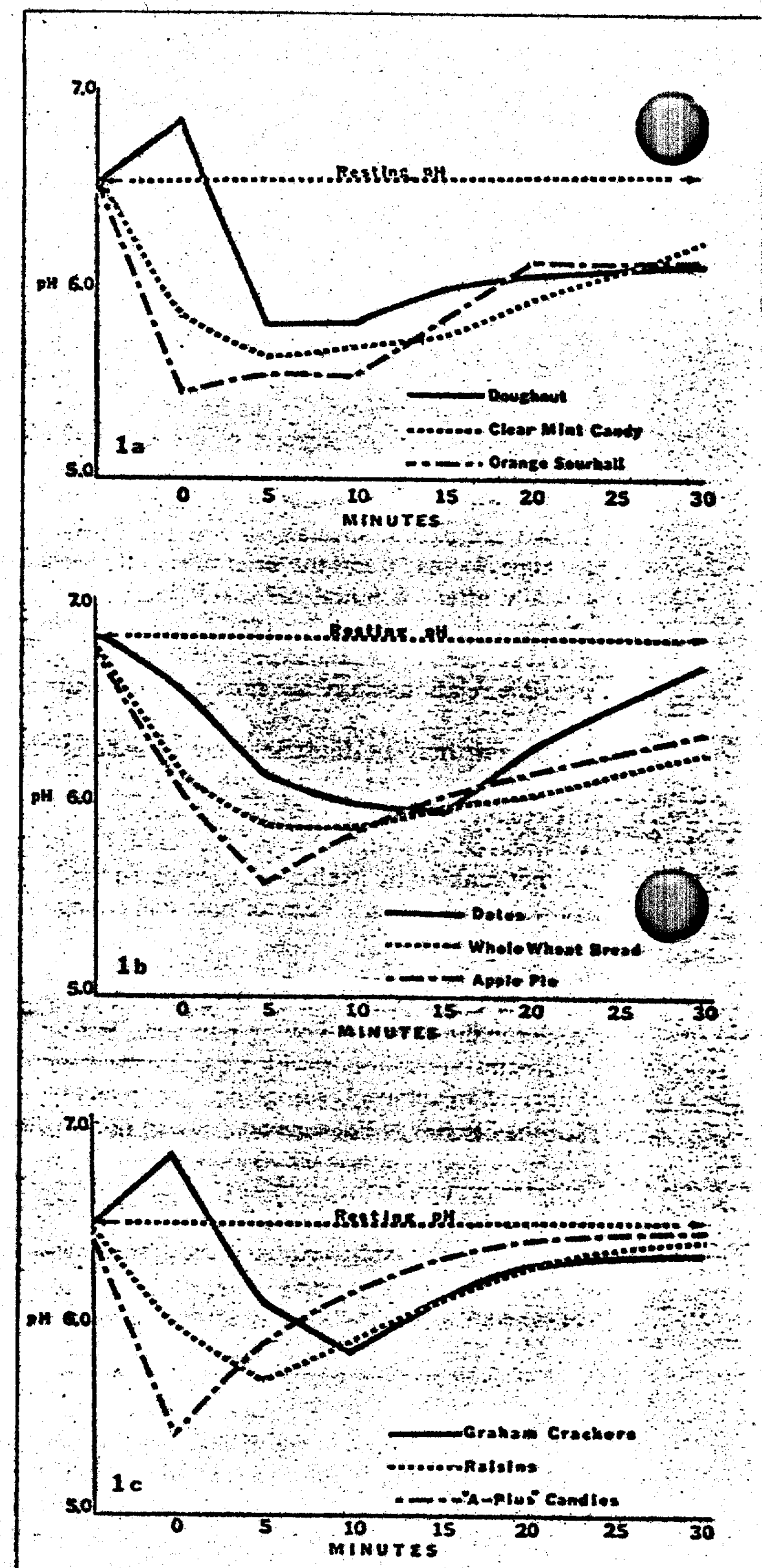

Fig 1 ▪ Types of plaque pH curves. Top, normal pattern. Center, prolonged pH fall (pH minimum rank lower than ΣD rank). Bottom, brief pH fall (pH minimum rank higher than ΣD rank).

Table 3 ■ Foods grouped by category and acidogenic potential.

Group	Beverages	Fruit	Bread, cookies, crackers	Cakes	Candies	
					Hard	Soft
1	Milk	Peanuts				Sugarless gum
2	Chocolate milk	Potato chips Apple	Bread & butter Graham crackers		Sweetees	Caramels Sugared gum Chocolate Licorice candies
3	7 up Coca-Cola	Banana	Sandwich cookies Crackers Bagel	Twinkies		Orange Giant Jellies
4	Apple juice Orange juice	Dates Raisins Cereal	Bread & jam Whole wheat bread Plain cookies	Doughnut Most cakes		
5	Coca-Cola (rinse)		Apple pie Chocolate grahams	Angel food cake	A-Plus Rock candy Clear Mints	
6					Sourballs Suckers	Ju-ju Toys

selected plaque pH curves for foods in the three categories based on discrepancies in ranking. Although the differences in the patterns of pH fall were not great, some of the expected patterns were seen, especially the delayed pH fall for type B curves and the brief fall in pH (occasionally after an initial pH rise), increasing to approach resting levels for type C curves. These discrepancies might be expected to modify any possible relationship of the rank numbers shown in Table 2 with the cariogenicity of the foodstuff. This point may be illustrated by consideration of two foods: lycasin candy was ranked 51st for pH minimum, but 9th for ΣD, whereas apple pie was ranked 35th and 49th, respectively. Clearly, neither ranking by itself can be expected to yield a wholly satisfactory index of potential cariogenicity.

Accordingly, the foods were grouped by inspection of the pH minimum data into six groups, of ascending acid-provoking potential, but with this adjustment: foods with type B curves were allocated to a more "cariogenic" group and type C foods to a less "cariogenic" group (Table 3). This table also categorizes food classes, and it is of interest to note the positions of the majority of hard and soft candies relative to each other, and in addition the relatively nonacidogenic nature of carbonated beverages compared with fruit juices and other foods.

■ *Food clearance studies:* An examination of the relationship between the pH changes in plaque and the rate of clearance of the food from the mouth is of interest. The clearance studies showed that, after 30 minutes, the amount of carbohydrate retained in the mouth was little more than the amount found by oral brushing and rinsing without food (Table 4). At 5 and 15 minutes, however, a variable amount of carbohydrate, greater than that at the baseline level, was found in the rinsings. The values for 5 and 15 minutes were closely correlated ($r=0.83$) and, therefore, it may be assumed that the amount of carbohydrate retained five minutes after consumption of a foodstuff is a useful index of its retentivity or clearance rate. Correlation coefficients between rankings for five-minute carbohydrate levels and pH minimum and ΣD values were calculated for the 48 foods that were tested both for plaque pH and retention and revealed a statistically significant correlation ($r_s=0.30$; $0.05>P>0.01$) between retention at five minutes and pH minimum, and a weaker correlation ($r_s=0.24$; $0.10>P>0.05$) between retention at five minutes and ΣD.

It is of interest to note the foods whose five-minute retention and pH minimum values did not give a significant correlation coefficient were those foods that provoked a larger pH drop than would be predicted from their five-minute carbohydrate levels (sourballs, suckers, 7 up, orange juice, apple juice, rock candy) and those foods that are retained in large quantities in the mouth but that do not give rise to a correspondingly low pH minimum value (chocolate, caramel, Orange Giant Jellies). The predominantly acid nature of the first group suggests that one parameter that must be included in an assessment of food cariogenicity is the pH of the food itself. For the second group, it may be postulated that foods that give rise to an excessively high concentration of sugar in the environment of the plaque exert an inhibitory effect on the bacteria and thus reduce the severity of the initial pH fall. The possibility remains that when the food is diluted to noninhibitory levels, delayed overall pH depression may be just as severe.

■ *Food saliva pH and carbohydrate concentration:* The contribution of the food pH and soluble carbohydrate parameters to the plaque pH changes was evaluated by analysis of the relationship between the pH minimum of plaque

Table 4 ▪ Mean carbohydrate (mg glucose equivalents) retained in mouth at 5, 15, or 30 minutes after food use.

Food (ranked for plaque pH minimum)	Mean carbohydrate, retained 5 min	15 min	30 min	5-min carbohydrate rank (lowest first)
Trident	1.5	2.0	1.8	1
Peanuts	4.9	3.3	2.6	4
Milk	8.4	3.7	2.5	11
Licorice Giant Jellies	Not done			...
Licorice Bites	6.5	4.6	3.8	7
Dentyne	5.0	3.9	3.1	5
Dark chocolate	24.6	8.1	3.5	40
Apple	11.4	4.3	2.9	14
Potato chips	12.3	4.9	2.5	17
White bread	16.1	10.0	3.6	26
Milk chocolate	19.0	6.8	3.0	33
Licorice Drops	15.8	8.2	3.0	25
Chocolate milk	7.4	3.8	1.9	9
Sugar-coated gum	17.9	5.3	3.4	30
Caramel	19.0	4.2	2.5	34
Bread & butter	16.1	7.7	3.8	27
Saltine crackers	18.7	6.8	5.2	31
Bagel	13.0	11.1	4.7	19
Banana	11.8	5.1	3.2	16
Sweetees	3.4	2.4	2.5	2
Whole wheat bread	15.5	6.9	3.3	23
Dates	11.7	5.0	3.0	15
Bread & jelly	21.8	6.1	4.7	37
Graham crackers	26.3	9.0	5.9	42
Orange Giant Jellies	49.8	10.1	4.2	47
Oil-sprayed crackers	23.8	8.5	3.7	38
Unsalted crackers	33.0	10.4	3.3	45
Coca-Cola (drink)	7.7	3.6	2.4	10
Twinkies	21.6	6.3	3.8	36
7 up	6.3	2.4	2.1	6
Doughnut	15.2	7.5	3.4	22
Pound cake	24.6	8.8	4.3	41
Cereal	14.6	6.5	2.7	20
Sandwich cookies	35.0	8.4	4.9	46
Apple pie	20.0	7.9	5.7	35
Apple juice	7.3	3.0	2.2	8
Astrofood	14.8	5.4	3.4	21
Sponge cake	18.8	6.0	4.2	32
Chocolate cake	25.7	10.6	4.9	39
Plain cookies	32.6	14.2	4.1	44
Rock candy (dark)	Not done			...
Rock candy (light)	9.4	2.7	2.4	12
Mint candy	31.9	9.4	2.5	43
Orange juice	3.6	2.8	2.1	3
Coca-Cola (rinse)	Not done			...
Raisins	16.8	5.7	3.0	29
Chocolate graham	10.4	3.4	2.8	13
Angel food cake	17.4	11.4	3.5	28
Raspberry sucker	Not done			...
Ju-ju Toys	75.9	10.7	3.6	48
A-plus	Not done			...
Orange sourballs	15.7	4.1	3.4	24
Grape sourballs	Not done			...
Cherry sucker	12.8	3.6	2.7	18

and the pH and the soluble carbohydrate content of the expectorated food-saliva bolus. The soluble carbohydrate levels determined on parallel samples of the snack foods were used to calculate the soluble carbohydrate values in the bolus and thus the carbohydrate environment of the plaque.

The relationship between plaque pH minimum and saliva pH is complicated by the likelihood that a more acid food will increase the rate of salivary flow and thus reduce the retention of food in the mouth. For examination of this complicated relationship, Kendall's partial rank correlation coefficient[9] was calculated for foods other than beverages, to determine the correlation between plaque pH minimum and five-minute carbohydrate retentions, holding the variable of food pH constant. This analysis resulted in a slightly higher correlation than was found when food pH was not taken into account; this indicates that the pH of the food did influence the relationship between plaque pH and food clearance. However, the main influence of food pH was not a direct effect on plaque pH itself, but rather an indirect relationship between food acidity and the rate of removal of carbohydrate from the mouth. This surprising result, although true for the average, may be less valid for individual foods of high acidity. Causal connections between food pH and carbohydrate retention may lie in the nature of the foods themselves (acid foods tend to have less carbohydrate) or in the effects of acid on the rate of flow of saliva as previously postulated.

The influence of the concentration of soluble carbohydrate in the bolus on plaque pH would not be expected to be linear, since inhibition of pH fall in plaque could result from both low and